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The Stability of the Avirulent Characters of Brucella Abortus, Strain 19 and Strain 45/20 in Lactating and Pregnant Cows

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McEwen (1940) demonstrated the instability of the avirulent characters of *Br. abortus* strain 45/20 in pregnant cattle. Subsequently, Edwards, de Ropp and McLeod (1945), during the course of their immunological experiments with 45/20, could not exclude the possibility that even in non-pregnant cattle it might alter in character and regain its former virulence. On the other hand, McEwen (1946) found no evidence that this strain became virulent when used for vaccinating non-pregnant cattle in the field. The stability of the avirulent characters of *Br. abortus* strain 19 has been confirmed by several American workers (Mingle, Manthei & Jasmin, 1941; Birch, Gilman & Stone, 1943; and Gerhard & Wilson, 1948), and in this country by Edwards, McDiarmid, de Ropp & McLeod (1946), who showed that this strain is harmless when injected into non-pregnant heifers and that the organism is not excreted in the milk during the subsequent lactation. Moreover, McDiarmid (1949) has shown that, in certain circumstances, pregnant cattle may be vaccinated with strain 19 with comparative safety.

The object of the work described in this paper was to study further the stability in cattle of both strains 19 and 45/20. Part I refers to the inoculation of lactating cows with vaccinal doses, whereas Part II describes the passage of each strain in large intravenous dosage through pregnant cattle.

PART I

MATERIAL AND METHODS

In general, the methods employed were similar to those described by Edwards, McDiarmid, de Ropp and McLeod (1946) and may briefly be described as follows:—

Experimental Animals.—Twenty-eight non-pregnant Ayrshire and Friesian cows negative to the agglutination test. These animals had already calved once and, with three exceptions, were lactating at the time of vaccination.

Vaccination.—The routine method of subcutaneous injection was used. Eight cows received 5·0 ml. of S.19 vaccine; eight received two separate inoculations of 4·0 ml. 45/20 vaccine at an interval of three weeks. The vaccines were obtained from the Ministry of Agriculture.

Agglutination Tests.—The scheme of Stableforth (1936) was used.

COLLECTION AND EXAMINATION OF MATERIAL

This has already been described by Edwards *et al.* (1946). Duplicate plates of bactotryptose blood agar were inoculated with samples of colostrum, cotyledon or milk and incubated aerobically and in 10 per cent. CO_2. This technique was checked by the culture of known *Br. abortus* strains under the same conditions. Guinea-pigs used for the biological tests were killed three weeks after inoculation to ensure the recovery of S.19 if this strain should be present.

EXPERIMENTAL PROCEDURE

The 28 cows were housed in the same dairy and grazed in the same fields and were divided into three groups, *viz.*:—

(1) 45/20 group—eight animals.

(2) S.19 group—eight animals.

(3) Non-vaccinated controls—twelve animals.

Services commenced two months after vaccination. Prior to parturition the following samples were taken:—

Milk samples at fortnightly intervals until the animal became dry. These were examined culturally in all cases and in addition samples from the vaccinated animals were tested biologically at monthly intervals.

Blood for the agglutination test 14 days after vaccination and thereafter at monthly intervals.

At parturition the following materials were collected:—

(1) Blood for agglutination test.

(2) Colostrum.

(3) Cotyledon or Cervical Swab.

For cultural and biological examination in the vaccinated groups and cultural examination only in the non-vaccinated group.

After parturition, in the subsequent lactation, milk samples from each cow were examined culturally every 14 days for a period of ten weeks and milk from the vaccinated animals was examined biologically at four and eight weeks. In addition blood samples for agglutination tests were taken from each animal at intervals of one month after calving.

Results

The results are shown in Table I and it will be seen that no evidence was obtained of the excretion of *Br. abortus* from lactating cattle vaccinated with strain 19 or 45/20. Moreover, no infection was demonstrated in the in-contact control animals.

TABLE I: THE BACTERIOLOGICAL EXAMINATION OF CATTLE VACCINATED WITH STRAINS 19 AND 45/20 AND NON-VACCINATED CONTROLS

Group	Pre partum milk samples		Maximum mean agglutination titre subsequent to vaccination and prior to parturition	Examination at parturition					Post partum milk samples	
				Mean blood titre	Colostrum		Cotyledon			
	Cult.	Biol.			Cult.	Biol.	Cult.	Biol.	Cult.	Biol.
45/20 (8 cows) ..	105 N	32 N	N	N	8 N	8 N	8 N	8 N	40 N	16 N
S.19 (8 cows*) ..	66 N	29 N	1 : 3,400	1 : 240	6 N	6 N	6 N	6 N	30 N	12 N
Controls (12 cows) ..	176 N	..	N	N	12 N	12 N	12 N	12 N	101 N	..

N signifies negative. .. signifies not done. * signifies one cow destroyed prior to parturition and one cow not pregnant.

PART II

Experimental Procedure

The scheme of the experiment was to inoculate each strain into a pregnant cow in a dose sufficient to produce abortion, recover the organisms in culture, then with as short a period as possible on artificial medium inoculate another pregnant animal. This procedure was repeated until both strains had been passaged seven times. After each passage an attempt was made to assess, both *in vitro* and in guinea-pigs, any departure from the recognised characteristics of the strains.

Materials and Methods

Cultures.—Both strains were received in the dry state from the Veterinary Laboratory, Weybridge, subcultivated on bactotryptose agar and a 48-hour subculture from this was used to inoculate the first passage animals. At abortion, cotyledon and/or foetal stomach contents were spread over blood-bactotryptose agar plates, incubated in duplicate in air and 10 per cent. CO_2 and, when possible, 50 colonies picked off. All of these were tested by the thionin blue method (McLeod, 1944) for the identification of S.19, then one half was used in phase determination by the thermo-agglutination method, and the other subcultivated and mixed to provide inocula for the next passage and for virulence determination in guinea-pigs. Thus the organisms grew *in vitro* twice between each passage.

Experimental Animals.—Fourteen cattle, four to six months in calf, from a herd consistently negative to the agglutination test, were used. Before and after inoculation, precautions were taken to avoid all risk of extraneous infection. Each animal was inoculated intravenously with approximately 20 times the recommended vaccine dose of S.19 in a volume of 50 ml. The mean viable counts were: S.19, 14×10^{11} organisms, and 45/20, 11×10^{11} organisms.

Assessment of Virulence in Guinea-pigs

It is known that strain 19, after inoculation in suitable dosage into guinea-pigs, produces neither splenic enlargement nor persistent agglutinins and may be recovered culturally from the spleen three weeks but not seven weeks after inoculation. Similarly, strain 45/20 produces neither agglutinins nor splenic enlargement and may be recovered seven weeks but not 15 weeks after inoculation. A virulent strain, on the other hand, causes persistent agglutinins in high titre, marked splenic enlargement and may be recovered in culture from the spleen up to six months after inoculation.

Accordingly, groups of 15 guinea-pigs were inoculated subcutaneously with each strain, *i.e.*, prior to passage and after recovery from each cow. The infecting dose in each case was calculated as one million organisms (average viable count: S.19, 85×10^4 organisms; 45/20, 77×10^4 organisms). Five guinea-pigs from each group were destroyed three, seven and 15 weeks after inoculation and the agglutination titre, spleen weight/body weight ratio (de Ropp, 1945) and the presence or absence of Brucella in the spleen determined.

Results

Table II shows the result obtained when *Br. abortus* S.19 is passaged in large intravenous dosage seven times through pregnant cattle. In so far as was determined the strain remained unchanged; it retained both its ability to grow in air and its accepted virulence for guinea-pigs; its phase remained smooth. Further, all of the 350 or so single colonies examined by the thionin blue test exhibited the specific reaction shown to this dye by S.19.

Strain 45/20, on the other hand (Table III), by the seventh passage had become a highly virulent CO_2 sensitive strain. Its "R" character, and consequently its inability to produce agglutinins, was lost between the second and third passage and the inoculation of further animals merely served to emphasise and perhaps to enhance, its altered virulence. Sensitivity to CO_2 returned more gradually. This was first observed after the third passage, when the organisms recovered showed a greater colonial size when incubated in CO_2 than in air. By the seventh passage, organisms incubated for 48 hours in 10 per cent. CO_2 were of normal size, whereas after ten days' incubation in air they were just visible to the naked eye.

Summary

1. Neither *Br. abortus* strain 19 nor strain 45/20 was recovered from the cyetic products of cattle vaccinated during lactation.

2. *Br. abortus* strain 19 retained its cultural and biological characteristics after seven serial passages in large intravenous dosage through pregnant cattle, while strain 45/20 on similar passage reverted to a fully virulent CO_2 sensitive strain.

Acknowledgment

We are indebted to the Director of the Field Station, Dr. W. S. Gordon, for his continued interest in this work.

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Table II: The Passage of *Brucella abortus* Strain 19 Through Pregnant Cattle

Passage	Abortion		Phase		Virulence for guinea pigs								
					3 weeks			7 weeks			15 weeks		
	Days	Titre	"S"	"R"	Ratio	Titre	*Br. ab.*	Ratio	Titre	*Br. ab.*	Ratio	Titre	*Br. ab.*
			25	0	0·14	1 : 40	+	0·14	1 : 20	—	0·16	N	—
1	16	1 : 1,280	25	0	0·12	1 : 76	+	0·12	1 : 4	—	0·15	N	—
2	25	1 : 2,560	25	0	0·17	1 : 34	+	0·17	1 : 6	—	0·12	N	—
3	41	1 : 5,120	24	1	0·18	1 : 84	+	0·11	1 : 6	—	0·13	N	—
4	42	1 : 320	25	0	0·17	1 : 24	+	0·11	N	—	0·12	N	—
5	32	1 : 2,560	25	0	0·13	1 : 38	+	0·13	1 : 4	—	0·11	N	—
6	30	1 : 5,120	25	0	0·12	1 : 52	+	0·13	1 : 10	—	0·12	N	—
7	24	1 : 20,480	25	0	0·11	1 : 18	+	0·15	1 : 6	—	0·12	N	—

N signifies negative in a dilution of 1 : 10.

Table III: The Passage of *Brucella abortus* Strain 45/20 Through Pregnant Cattle

Passage	Abortion		Phase		Virulence for guinea pigs								
					3 weeks			7 weeks			15 weeks		
	Days	Titre	"S"	"R"	Ratio	Titre	*Br. ab.*	Ratio	Titre	*Br. ab.*	Ratio	Titre	*Br. ab.*
			0	25	0·12	N	+	0·15	N	+	0·13	N	—
1	12	1 : 20	0	25	0·12	N	+	0·16	N	+	0·13	N	—
2	37	1 : 20	0	10	0·12	N	+	0·15	N	+	0·15	N	—
3	47	1 : 20	10	15	0·14	1 : 128	+	0·28	1 : 1,696	+	0·53	1 : 10,240	+
4	37	1 : 2,560	25	0	0·16	1 : 256	+	0·41	1 : 6,672	+	0·65	1 : 74,242	+
5	24	1 : 10,240	25	0	0·13	1 : 432	+	0·49	1 : 960	+	0·47	1 : 14,366	+
6	10	1 : 1,280	25	0	0·17	1 : 352	+	0·51	1 : 8,840	+	0·56	1 : 19,280	+
7	9	1 : 2,560	25	0	0·16	1 : 352	+	0·56	1 : 4,416	+	0·37	1 : 13,312	+

N signifies negative in a dilution of 1 : 10.

Virus Diseases in Dogs*

By

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GLASGOW

When I was asked by your Secretary to read a paper on this subject I pointed out that I had no special qualifications to give an original paper but I expressed my willingness to review present knowledge on the subject as an introduction to a general discussion. I understand your Society draws its members from many parts of the country and I do think that at such a meeting as this, there is much clinical knowledge that could be pooled, particularly in relation to the epizootiology and varieties of disease that occur in different parts of the country.

In *The Veterinary Record* of a fortnight ago J. R. M. Innes discusses the relationship of distemper virus to the canine encephalopathies. He pays tribute to the notable work of MacIntyre, Trevan and Montgomerie and makes an excellent critical review of publications up to date. He stresses that no disease is recognised until it is found by looking with great care, an opinion I heartily underwrite. Many diseases exist over wide areas unrecognised and it is only when someone establishes the presence of a particular disease and describes its clinical manifestations in one country that it becomes recognised as having a wide distribution. It is worth stressing that the Burroughs Wellcome team's excellent work was not dependent on any new method of investigation and the fundamental knowledge that made it possible has been available for the last ten years. The essential requirement for the investigation of virus disease is co-operation between field workers who are working with naturally occurring diseases and research workers who have opportunities for pathological investigation and the use of animals of the same or related species for experimental work. Obviously these conditions never normally can apply in the investigation of serious human disease and for this reason veterinarians can play an important part in the accumulation of knowledge of the virus-host relationship and in elucidating their many problems.

I intend reviewing canine virus diseases as they occur in this country and as they have been reported abroad, stressing particularly the clinical aspect and attempting to describe a reasonable method of differential diagnosis and an account of symptoms which would permit recognition of clinical entities at present obscure. As practitioners, our main contribution should be careful selection of material from isolated outbreaks in kennels, or in remote or isolated districts, particularly where there is some knowledge of the dogs' immunological backgrounds. I once had an opportunity of studying cases in an outbreak of distemper from one of the small islands in the Hebrides which had been free of infectious disease for years ; with present-day facilities such an opportunity would be exceedingly welcome.

A feature that became very obvious at the last N.V.M.A. Conference was the variation in symptoms in different parts of the country. I myself was dealing with a hard pad outbreak in Glasgow that differed markedly in its clinical manifestations from one in Edinburgh only 50 miles away ; and the syndrome described to me by colleagues in the South of England appeared to be part of a different disease.

The limiting factor to unlimited investigation at the moment is probably the small number of research workers engaged on the subject—it is certainly not lack of material. Fortunately, the ranks of investigators have been augmented by the establishment of the Canine Research Station of the Animal Health Trust and I understand virus disease investigation is on their programme. There are many lines of investigation that suggest themselves, not the least important of which would be random sampling of blood and routine complement fixation tests. An important advance was made recently by Haig[1] at Onderstepoort when he succeeded in cultivating Green's distemperoid virus on chorioallantois. This should open up many possibilities for new methods of prophylaxis and investigation. I understand it has been impossible to establish Green's virus in this country, partly no doubt due to transport difficulties, but this discovery should make the matter much easier. B.E.A. could bring an egg in 36 hours.

In the early part of the century there was much conjecture as to the actual cause of distemper, but few people at that time considered that it might be a group of diseases. Carré's work demonstrating that a virus was responsible, was not entirely accepted in this country and it was only when Laidlaw and Dunkin[2] (L. and D.) completed their work that the virus theory became universally accepted. The varied clinical manifestations were presumed to arise from a host of secondary invaders. L. and D. succeeded in eliminating these after serial passage but were satisfied that the injection of their prophylactic gave the animals sufficient immunity to natural disease and so prevented the secondaries from establishing themselves. They expected that their experimental disease would be milder because of the absence of secondary pathogens and when experimental distemper proved to be a comparatively mild disease they were not surprised. Most practitioners reluctantly accepted this explanation of the disparity in virulence between experimental and street distemper, a difference in mortality of 5 to 10 per cent. compared with 50 to 100 per cent. L. and D.'s work was so excellent that veterinarians here accepted their entire findings and this included their dictum that distemper in this country was caused by one virus. As far as I can discover, only six strains of the virus were investigated and these were selected by Dunkin from field cases which he considered to be typical of distemper. The results in the field with their prophylactic were excellent at this stage, the increased survival rate in some hound kennels leading to overproduction ; but the breakdowns were rarely explained satisfactorily. It has often been the case that people who have made an excellent contribution to scientific advance frequently have delayed further progress in their particular subject by a dogmatic pronouncement which, while true in their limited experience, was not of universal application, and I think this indeed happened here.

Perdeau and Pugh (P. and P.) in 1930[3] described a series of distemper-like illnesses in which a number showed a micropathology very different from that described by L. and D. They concluded that in this demyelinating encephalitis the distemper virus stood in the same relationship to the brain lesions as some virus diseases were reputed to do in relation to subacute demyelinating sclerosis in man. When one reviews the earlier pathological work on distemper encephalitis it becomes obvious that many cases diagnosed clinically as nervous distemper were obviously not caused by L. and D. virus. As early as 1912, Cerletti's[4] description was much more akin to P. and P.'s description than to L. and D.'s and during the last ten years there have been many descriptions in European and American literature of demyelinating encephalitic disease in dogs. Cordy in 1942[5] correlates pathological and clinical findings in naturally occurring and experimental disease and from his description one can recognise several clinical entities that occur in this country.

It has always been the case that a successful new prophylactic or a new specific therapeutic agent permits the further differentiation of a group of diseases which previously had been ascribed to a common cause. Distemper prophylaxis was no exception and many apparent breakdowns in immunity in vaccinated dogs were shown to be free from L. and D. virus when injected into ferrets and therefore probably had a different cause. Because of the routine adopted of challenging the immunity of such ferrets as failed to react within ten days to the possible presence of virus in suspected tissue, with an injection of known L. and D. virus, many of the cases which might have developed disease as a result of the first injection did not have this opportunity, or when they did, this reaction was presumed to be caused by the second injection. Over a period of years this could have obscured hundreds of results and hard pad (H.P.) virus may have been present in this country for many years, but the ferret test normally carried out was not likely to reveal it. If what I suspect happens in dogs when one virus is injected into a case harbouring a previous one, also happens in ferrets, then probably they would rapidly succumb to the challenging dose of L. and D.

Most practitioners, myself among them, were satisfied that if a dog showed chorea it could be accepted as evidence of L. and D. virus infection, past or present, and even in the exceptional case where there was no history of febrile illness it was presumed that the infection had been sub-clinical. This belief was supported by the fact that L. and D., using bacteria-free virus, had produced disease in which chorea ultimately developed. Green[6] in America, investigating distemper-like disease in foxes, proved that there were two clinical entities, distemper due to L. and D., and fox encephalitis due to a virus transmittable to dogs but not to ferrets. I do not wish to mutliply references indefinitely, for there is much further evidence in the published literature of the diverse nature and cause of so-called distemper. The theory that varying secondaries could cause many clinical manifestations, while still true, could not explain all disparities and I think the profession was more than ready for the paper by MacIntyre, Trevan and Montgomerie[7].

Before the Southport Congress I had submitted a series of cases with clinical histories to Dr. Montgomerie and his colleagues. Material sent usually included sterile brain and spleen and formolised bladder, brain, lung, and sometimes, pads ; lately whole

* Presented to the meeting of the Society of Women Veterinary Surgeons, held at the Royal Veterinary College, London, February 26th, 1949.

carcases. They carried out extensive microscopical examination and usually injected ferrets and dogs, and I received reports on 16 of these prior to the Congress.

I propose to attempt to classify the various virus diseases in the light of such information as has been published up to date, combined with the evidence which arose out of the investigation of this series. Ten of these cases were attributed to H.P. virus (Type I B); two to Type II, demyelinating disease not affecting ferrets; three to Type III. In one case there was no material available for injection, but microscopical appearance, while inconclusive, suggested Type I B (L. and D.).

I include in this classification several conditions which I have not observed personally but which I feel sure are occurring in this country. Rubarth's[8] recently published work on the occurrence of *Hepatitis contagiosa canis* in Sweden covers the last 15 years and he has established the existence of a clinical entity which could quite easily be missed in practice. More recently Coffin[9] has demonstrated the natural occurrence of this disease in the U.S.A. and I feel it is quite possible that the disease exists unrecognised in this country. There seems to be little doubt that this disease is identical with Green's fox encephalitis. I can recollect distinctly cases with a history similar to that described by Coffin and I look forward to investigating the next one. From what has been said I think it is permissible to assume that much so-called clinical distemper occurring during the last 30 years in the new and the old world has been due to some cause other than L. and D. virus. In my practice this is much less common than formerly.

Distemper: L. and D. Virus

I think one must refer to this as L. and D. and not as Carré, as is usual in American publications, as at this date it is impossible to establish whether Carré worked with L. and D. virus or some other virus: such evidence as is available suggests that it was not L. and D. One must consider this as two distinct clinical manifestations: (*a*) distemper caused by L. and D. virus administered experimentally after serial passage which had rid it of associated secondary organisms and is a comparatively mild disease; (*b*) distemper caused by L. and D. virus (street or field) and which has superimposed, because of secondary organisms, many varied signs.

The following features follow in experimental inoculation with L. and D

A catarrhal fever with an incubation period of about four or five days and a characteristic diphasic fever curve. During the first 24 hours there is a watery discharge from the eyes and nose which becomes faintly purulent (this stage is very highly infectious). There is acute conjunctivitis and crusts gather at the angle of the eyes. The ocular symptoms usually diminish in one week but keratitis supervenes in a small percentage of cases. At the outset of the disease many dogs vomit and refuse food but appetite returns when the fever departs; diarrhoea is a feature throughout the disease, the movements are offensive, slimy and streaked with blood and the diarrhoea may continue for some days after the fever has disappeared. As might be expected, there is a great deal of loss in general condition. During the second stage there is a slight cough and occasionally at the beginning and often towards the end of this stage nervous symptoms appear, L. and D. estimated, in about 10 per cent. of cases (fits and epileptic fits). MacIntyre *et al.* record a much smaller percentage of fits than this in the course of immunising many dogs against distemper. Chorea usually appears towards the end of the illness.

Microscopical examination of the brain revealed small, irregularly distributed, areas showing degeneration in brain cells and small haemorrhages. There were collections of round cells in the cortex and around the ependyma.

Experimental inoculation.—Ferrets in ten days developed coryza, vesicles and pustules round the mouth and usually died the fourth day. Experimental inoculation by any route into dogs produced ordinary distemper. No neurotropic form of the virus has been demonstrated in this country, although in France Goret describes a neurotropic one. Certainly, injection of known virus into the brain produces the usual syndrome of *distemper, not encephalitis.* One very significant feature of L. and D.'s experiments with dogs reared in isolation is the fact that chorea developed in a proportion of cases following experimental injection with L. and D. Many people have postulated that the chorea is caused by the activation of a virus already present. If this were so in their case the virus must have been transmitted through two or three generations of dogs from the original stock where, of course, it may have been latent.

Distemper Due to L. and D. Virus (Street or Field)

The symptoms are similar to those described above. There is more profuse nasal discharge and the respiratory symptoms are much more severe, being of a broncho-pneumonic character and running a ten-day course. The initial fever curve is still diphasic. In many cases skin lesions characterised by pustulation are present early in the disease but these can occur apart from distemper infection, with distemper infection, or in distemper-immune dogs; and when they do occur in distemper-immune dogs they give no protection against distemper. *Bronchisepticus* infection was responsible for some of the lung lesions and streptococci frequently were involved in pneumonia. In some outbreaks keratitis is a regular feature and ulceration of the cornea is fairly frequent. In some outbreaks, again, the percentage of nervous complications is much higher than in the experimental disease and certainly this is the case with posterior paralysis, although almost invariably I found such cases recovered provided the condition was not complicated by chorea and provided the animal was given sufficient time.

When a dog starts convalescence the nose has the appearance of burnt cork and I consider the mode of healing is characteristic in this disease. In H.P. disease during the convalescent period the nose starts to peel and when this is complete there is rapid and uniform restoration of the mucous glands and smooth cool nose. L. and D. disease healing commences at the labial margin and proceeds slowly over a period of four to six weeks. Halfway through this period the lower half of the nose is glistening and moist and the upper part is still dull and dry. I think this is probably typical of L. and D. virus. I have seen this disease occur in a pregnant bitch and one of the puppies showed chorea in one limb within 48 hours of birth. When an outbreak occurs in a kennel I have rarely seen suckling puppies develop the disease unless the mother succumbs to infection. It may be that they receive some protection from the colostrum but when the mother does develop the disease the pups usually die within a week, after showing a few catarrhal symptoms. If a bitch nursing pups becomes infected with "hard pad" virus the pups usually show no catarrhal symptoms but take epileptic fits and die within a few days. Some even develop hard pads. Puppies between six and ten weeks frequently show only bowel symptoms at first, and appear in many cases to escape respiratory symptoms. If the condition is protracted the second teeth on, or soon after, eruption show dentine hypoplasia or "canker mouth," the condition described by Mellanby as being caused by vitamin D deficiency. My experience in actual practice suggests that such a deficiency nearly always arises as a result of virus infection—so much so, that I usually advise people that it is probably unnecessary to carry out distemper inoculations in such a case.

Diagnosis.—I consider the diphasic curve very important but one is likely only to discover this when dealing with an outbreak in a kennel. When disease breaks out all the young susceptible dogs have their temperature taken morning and evening and this is sufficient to establish the characteristic curve. If ferrets are in contact, as frequently happens at a gamekeeper's, they rapidly develop the disease.

Distemper Due to Green's Distemperoid Virus

This usually produces a slight febrile reaction in dogs although in ferrets it produces a rapidly fatal disease. According to French reports by Goret[10] the reaction has some bearing on the size of the dose and it has been found that the dose (25 mg.) normally given in U.S.A. for prophylaxis produced typical distemper in 10 per cent. of cases, and 7 per cent. with doses of 15 mg. and 2 per cent. with doses of 10 mg., with a percentage of these developing nervous complications with fatal results. So far as I know, this virus has never been successfully brought into this country.

Recently Goret has suggested that the distemper occurring in France meantime and during the last ten years may owe its virulent character to the importation of Green's distemperoid vaccine which he considers a highly virulent pathogen. (He presumes that after the first passage in dogs it regains its virulence.)

Epidemic Encephalitis, or Epizootic Encephalitis, or "Hard Pad" Disease (MacIntyre et al.: Group I B)

There has been much speculation as to whether this is a new disease or whether the typical syndrome of hard pads was missed because it was not looked for. I think the disease is probably not new but that there has been some mutation in the virus which permits of a more regular and well defined appearance of hard pads in the course of the disease. I distinctly recollect dogs many years ago with deep fissures in the pads and later peeling, but I cannot recollect seeing what we now recognise as "hard pad" disease. There is even more speculation as to the antigenic relationship between this virus and the L. and D. virus. I have stated elsewhere that I consider that a dog that has passed through a natural attack of distemper is probably immune to "hard pad" virus and I feel that dogs that have been inoculated with L. and D. vaccine have a better chance of recovery than those uninoculated. Street or natural distemper in my district, I suspect, for many years has been of the "hard pad" variety and if this were so and L. and D. distemper had not

appeared naturally in my district for some time, then I may have to change my conception of their antigenetic relationship. "Hard pad" disease is characterised by a lengthy incubation period—probably a fortnight in dogs, anything up to a month in ferrets. The disease is sometimes ushered in with fever but the temperature may be normal or subnormal. There is no diphasic fever curve. The discharge from the eyes and nose is scanty and watery. There is some swelling of the lymphatic glands in the throat and neck. Severe diarrhoea and the expulsion of offensive watery faeces is a frequent symptom: in the early stages the movements are liquid and offensive but not necessarily frequent. There is offensive breath, the appetite is poor and muscular tremors are common. In some outbreaks respiratory symptoms are common. There is a broncho-pneumonia lasting eight to nine days, cough, fever and accelerated respirations, and diminished appetite. After apparent recovery a relapse is usual in three to four days when further respiratory symptoms develop—respirations are accelerated. Auscultation and percussion reveal very little and in fatal cases consolidation ensues about 24 hours prior to death, during which period the pulse assumes a running-down character.

The hardening of the pads can happen at any time within the first three weeks of the illness, occasionally later, and cases that recover peel from the pads and nose in a characteristic manner. In some outbreaks the disease is ushered in with several attacks of hysteria and there may be no further symptoms for weeks until the animal develops chorea, paralysis and fits, possibly epileptic. I think if this virus is a mutation, one of its main characteristics is a further capacity for variation or further mutation. In an outbreak which ultimately proved to be due to "hard pad" virus more than half the dogs failed to develop hard pads. Suckling puppies developed epileptic fits, some degree of hard pads and died. I think the highly infectious stage must be near the beginning of the illness. I have a case at the moment, an adult bitch suffering from "hard pad," showing nervous complication, and she has been in daily contact with one bitch and five puppies, none of which have shown any obvious symptoms. She herself probably acquired the infection from two dogs in the neighbouring house which died of "hard pad" disease. Either the bitch and her puppies were immune or they developed a sub-clinical form of the disease and are now immune: a blood test would be invaluable to demonstrate what has actually happened. The factor which protected them may, of course, have been a genetical one. I have never known a dog which had passed through a natural attack of "distemper" which developed "hard pad" disease. Self-mutilation occasionally occurs in this disease. Experimentally, ferrets develop a disease characterised by fever and hard pads any time between the tenth and 32nd day. Dogs develop only a febrile reaction.

Pathology.—The brain lesion is a demyelinating encephalitis, particularly in the cerebral peduncles and folia; when elsewhere —submeningeal or subependymal—glial proliferation, swelling and proliferation of the vascular endothelium, sometimes with histiocytic cuffing, intranuclear inclusions in the glial cells and histiocitis occur; but MacIntyre *et al.* have never seen inclusions in the nerve cell. Cytoplasmic inclusions (acidophilic) occur in alveolar and bronchial epithelium.

Experimentally, there is little immunological relationship with L. and D. virus.

MacIntyre et al.: Group 2 Disease

This is a disseminated demyelinating encephalitis in dogs which cannot be produced in ferrets. This may be "hard pad" disease after the "hard pad" virus has ceased to be present owing to the long duration of the condition. The disease is slow in developing and can affect dogs of all ages. Catarrhal symptoms, dry cough, enlargement of lymphatics may be present in the early stages, convulsions are usual, also paralysis, chorea and ataxia. MacIntyre, etc., consider circling to one side and bumping into objects a characteristic sign. Bodily condition is fairly good and death is usually preceded by coma. Further investigation of a few selected cases of circling dogs should clarify this condition.

Pathology.—Similar to "hard pad" virus, demyelination, gliosis, etc., but no ependymal inclusions and no inclusion bodies in the lung. MacIntyre *et al.* experimentally produced symptoms in one dog. In my series I had two possible cases of this type but clinically they were not characteristic. Circling was not a feature. One of my cases proceeded to mutilate an area of the abdominal wall which appeared to be associated with a chorea twitch. Serial section of the cord revealed a lesion in the posterior horn.

Disseminated Encephalomyelitis (MacIntyre et al.: Group 3)

This disease, usually affecting older dogs, is characterised by the insidious onset of nervous symptoms. As a rule, there is no history of catarrhal disease. The dog shows a change of nature, becomes dull, is frequently ataxic. He wanders about, frequently gets into a corner and appears to be incapable of action which would extricate himself. He ceases to show affection, takes frequent epileptic fits, loses interest in food but will eat if the food is put in or near his mouth. In my series one of my cases had been inoculated six months previously. Rest was taken only if the dog was placed in a recumbent position. He showed the usual symptoms, failed to recognise his owner, wandered about aimlessly. He tended to circle. The dog was not vicious but would snap if handled too much. I think this condition could be recognised clinically.

Pathology.—Cuffing of the blood vessels with small cells, mostly lymphocytes, in the perivascular space. No thickening of the blood vessel walls, no inclusions, no demyelination.

Experimental inoculation usually unsuccessful. In my series the experimental result was inconclusive. There was some evidence of reaction on the part of the ferret and in one case a slight elevation of temperature in a dog eleven days after.

Hepatitis Contagiosa Canis or Fox Encephalitis

By 1929 Green had established that much disease among foxes previously attributed to distemper virus was due to an unrelated virus with endothelial affinities. He found dogs were susceptible and always died within seven days. There were endothelial intranuclear inclusions. Ferrets were not susceptible to this virus. In Sweden, Rubarth has investigated 190 cases and carried out experimental work on a distemper-like illness which he calls *Hepatitis contagiosa canis*. Some of these cases developed nervous symptoms and there appears to be no doubt that the condition is identical with fox encephalitis. Coffin describes four cases in U.S.A. None of these showed nervous symptoms. It is very likely that this disease occurs in this country but one must appreciate that the well-recognised train of disease symptoms of a virus disease in one country does not necessarily correspond with what might be seen in another country. I append a brief list of signs. Rubarth describes apathy, sudden violent convulsions, great agitation, tonic or clonic spasms of the extremities—head and neck— and paresis of the hind quarters, occasional blindness and corneal opacity. The tonsils are red and swollen. The disease is spread by contact and in cage-to-cage infection. In a series of a hundred random blood samples 70 per cent. reacted. Many animals are resistant to experimental infection and many experimentally infected dogs recover (unlike Green's experience). Coffin in U.S.A. describes malaise, fever, soft gagging cough, watery discharge from the conjunctiva, tonsils swollen and red, loss of appetite, vomiting, collapse and death, although he had two recoveries with penicillin.

Post-mortem examination shows tonsillitis, liver dark-red to purple (sometimes mottled, grey areas from the size of a pinpoint up to 3 mm.). Gall bladder opaque.

Post-vaccination (Distemper) Encephalitis

I do not know whether the development of encephalitis in some cases following distemper inoculation might not be of the same nature as post-vaccination encephalitis occurring in humans. I understand that such an encephalitis (a disseminating encephalomyelitis) does occasionally follow the use of anti-rabid injection in dogs.

A severe perivascular round-celled cuffing present, chiefly in the grey matter. Glial clusters.

Toxoplasmosis (MacIntyre et al.: Group 4)

This is not a virus disease but I include it here for differential diagnosis.

MacIntyre reports one dog, although several have been reported in American literature, and a case in a ten months dog in Sweden. The symptoms described were high temperature for two weeks, temperamental changes and fits. *Post-mortem* examination showed a large area of necrosis in the medulla. Many toxoplasms were present, single and in clusters—pseudocysts. In humans diagnosis might be established by examination of the retina where there are widely scattered inflammatory and degenerative lesions.

Lymphocytic Choriomeningitis

The causal virus of this disease has been found in the C.N.S. of dogs but as far as is known, it does not cause disease.

This disease virus may be only of academic significance but it may be important in investigation.

General Observations

When one considers the virus-host relationship it seems unbelievable that so many viruses should have survived. I think there probably have been occasions when a virulent virus such as the 'flu virus of 1918 which swept over the entire globe, died out because it left in its wake a completely immune population and there was no mechanism for its survival pending the development of a new susceptible population. Some authorities consider that the normal mutation rate in viruses is very much greater than in bacteria and it seems likely that new epidemics originate in this way. A virus develops some new characteristic which allows it to establish itself in a new host (probably a species related to its

previous host with which it had established a balanced or almost commensal relationship) and it may become highly pathogenic. In nature it may be that viruses reasonably adapted to individuals of a species have an opportunity of developing epidemic disease when seasonal changes cause the animal to depart from a solitary existence and become gregarious; for instance, the normal behaviour pattern of wolves. Certainly the practice of taking in foxhounds from walk appears to repeat the natural pattern and we have all seen the epidemics which arise. The normal survival of distemper virus probably follows this course. There was an interesting French publication by Goret and Mery[11] in 1947 which dealt with four outbreaks of clinical distemper in isolated areas where it was considered that infection from dogs could not possibly have originated the outbreaks and the authors came to the conclusion that the disease probably originated in the small wild carnivores of the country (stoats, weasels, martens, etc.). All ages of animals were susceptible. Symptoms—hyperpyrexia, dysentery, encephalitis. Mortality, 30 per cent. *Post-mortem* examination—acute hepatitis, liver was very large; nephritic gangrene and Stuttgart syndrome; no pulmonary lesions. In the last three outbreaks injection of brain emulsion caused disease in ferrets (incubation period not stated).

Observations of outbreaks of disease in zoological animals by Armstrong and Anthony[12] have frequently demonstrated that L. and D. virus was responsible for distemper diseases in the Canidae (dog, ferret, red fox, eastern fox, kit fox, racoon dog, and Australian dingo). The administration of anti-distemper serum controlled the epidemics. This virus never affects the felidae.

Burnet considers that in perfect adaptation between host and parasite no inconvenience is caused and that the relationship has probably been longstanding. Such perfect adaptation because of the invisible nature of the organism would be impossible to demonstrate, as, until recently, the only method of recognising the presence of such an organism was by the effect it produced in the host. Occasionally such a non-apparent virus has been demonstrated by accidental means, as when a monkey bit a ruling monarch, and transmitted to him a virus which had caused the monkey no inconvenience but which ultimately killed the king. Once techniques are developed the electron microscope might reveal the presence of many viruses in the cells of the body, some possibly beneficial to the host, or even important integral factors in the innumerable enzyme systems of normal metabolism and that these viruses would only occasionally go berserk and cause disease. These enzymes are proteins and some contain nucleoproteins and must, by chemical and evolutionary standards, be related very closely to viruses. It has been suggested that in our group of distemper diseases chorea may be due to a non-apparent virus which lights up because of the introduction of another virus. Unfortunately the pathology of chorea has not been completely worked out. I would like to issue a warning here that the term chorea in human medicine describes a condition which is different from veterinary chorea (myoclonus). This latter has been attributed to an increase in the neuroglia round the cells in the anterior horn but I do not think the last word has been said with regard to its pathology. Recoveries (spontaneous or otherwise) which do occasionally occur would be difficult to explain. The lesions in human chorea are associated with the corpus striatum. I think the pathology of chorea could be elucidated fairly easily if we practitioners selected material from the C.N.S. for micro-pathological examination from cases of chorea affecting only single muscles or small groups of muscles and supplied an adequate case history for correlation.

In investigating virus diseases it must always be remembered that in any epidemic there is probably a large percentage of subclinical cases. Rubarth, investigating fox encephalitis, demonstrated by complement fixation tests that many dogs showed evidence of past infection without any history of previous illness. He concluded that subclinical cases were probably the rule. In human medicine it has been shown that most cases of poliomyelitis remain subclinical without the development of nervous symptoms. Occasionally an extrinsic factor brings to light that the virus is widespread. There was the well known case in the U.S.A. where three children developed acute poliomyelitis following tonsillectomy; subsequent examination showed that most of the affected children's companions were harbouring the virus.

With regard to MacIntyre's *et al.* classification, *re* Group II, I think it is quite possible that this may be caused by H.P. virus without the development of hard pads. In one outbreak I studied (later proved to be H.P.) many animals did not develop the characteristic pads. One of my cases which B.W. classified as Group II had been ill for over two months and the virus may have disappeared by the time the dog was destroyed. The absence of virus as demonstrated by ferret inoculation is the main reason for separating this disease from H.P. and if the protracted nature of the case is responsible for the absence of the virus then we are left with two major distemper diseases (1) H.P., (2) L. and D., and with several encephalomyelitic diseases. At the moment there is no satisfactory explanation for the anomalies in experimental work with H.P. virus. Injection of ferrets produces the disease but in dogs gives only a febrile reaction.

Green modified L. and D. virus by passage through ferrets and it produces an active immunity when injected into susceptible dogs. He also claimed that it could be used as a therapeutic in the early stages of the disease and explained this by the interference mechanism or cell blockage. He considered that the comparatively innocuous Green's virus blocked the cells from the more harmful distemper virus and that this for lack of a suitable nidus died off—a kind of scientific musical chairs. There is another phenomenon which has puzzled me very much and it may possibly be another manifestation of cell blockage. Sometimes a dog undergoing immunisation with vaccine-virus is presented for its second inoculation with a history of not being 100 per cent. fit and if clinical examination fails to reveal any abnormality the practitioner will probably consider it justifiable to administer the virus. We have all seen a proportion of these cases develop chorea or fits within 24 hours. I think such cases are suffering from subclinical virus infection other than L. and D. and as the primary virus is probably blocking most of the visceral cells the large dose of L. and D. virus distributes itself in the nervous system with disastrous results. Green describes a rather interesting phenomenon in fox encephalitis called temporary inactivation. He injected fox encephalitis virus mixed with hyper-immune serum; the latter appeared to hold the virus for five weeks and only after that period did disease develop. I think there is some evidence that this occasionally happens after serum virus inoculation (L. and D.).

Differential Diagnosis, Etc.

I realise that at this stage any attempt at complete classification can only be tentative, but even so, I wish to give a short section on differential diagnosis and in each of the diseases mentioned I have selected outstanding features which while not pathognomic, are useful pointers. I would also like to indicate procedures that might be developed to aid early diagnosis to assist in the administration of specific remedies. Methods of early diagnosis are very important and probably the most important requirement at the moment.

(1) L. and D. DISTEMPER.
- The diphasic temperature curve.
- Coryza and crusting round the eyes.
- Vomiting at the onset.
- Characteristic nasal appearance in convalescence.
- Epileptic fits occasionally at onset of second fever and often at end of the same fever.

Methods of Diagnosis which might be developed:
- Complement fixation.
- Examination for cell inclusions in cells scraped from conjunctiva or tongue, possibly in bladder cells centrifugalised from urine.
- Ferret inoculation—10-day incubation period.

(2) H.P.
- Insidious onset, occasionally ushered in with hysteria.
- Eyes slight catarrh, rarely profuse discharge.
- Hard pads at seven to 21 days.
- May be no fever; in fact, if catarrh is present, without fever, the disease is likely to be H.P.
- Respiratory catarrh is slight in the early stages.
- Offensive diarrhoea (sometimes infrequent) and bad breath and tremors.
- Late development of pneumonia with tendency to relapse after apparent recovery.
- Increased respirations.
- Running down character of pulse.
- Recurrence of fits, etc.

Possible Methods of Diagnosis:
- Complement fixation.
- Scrapings of tongue and bladder.
- Ferret inoculation.
- Centrifuge samples of urine to examine for inclusion bodies. In Sweden reports indicate that 90 per cent. of dogs suffering from distemper disease showed inclusions.

(3) GROUP II: I do not know of any characteristic syndrome but MacIntyre *et al.* consider circling to be characteristic. The dog remains in good bodily condition; there is little catarrh and no pneumonia.
- Protracted Course.

(4) GROUP III:
- Often adults are affected.
- Insidious onset.
- No catarrh.
- Dullness.
- Aimless wandering.
- Fits, paralysis, ataxia.
- No chorea.

(5) GROUP IV (TOXOPLASMOSIS):
Fits, fever, and changed temperament following a 14-day illness.
There may be lung lesions due to miliary distribution of the parasite.

Diagnosis:
Animal inoculation.
In humans the opthalmascope is useful in diagnosing the granuloma on the retina.

(6) CONTAGIOUS HEPATITIS FOX ENCEPHALITIS:
Type I (Coffin).
Fever, soft cough.
Slight discharge from conjunctiva.
Tonsils red and swollen.
Loss of appetite.
Vomiting, collapse and death.
Type II (Rubarth).
Apathy.
Sudden violent convulsions.
Great agitation.
Spasms of the extremities.
Blindness.
Corneal opacity.

Possible Methods of Diagnosis:
Post-mortem examination—liver dark red and purple with yellow areas.
Gall bladder opaque.
Endothelial inclusions.
Green's anterior chamber test in four and ferret test inconclusive.

(7) POST-VACCINATION ENCEPHALITIS:
Here the history should be some guide.

Although I have omitted all mention of treatment, I yet have to apologise for the length of this paper, which I feel could not have been shortened and remain in the least adequate in dealing with a subject of such complexity.

Acknowledgment.—I wish to acknowledge much help from Dr. Montgomerie and his colleagues.

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Discussion

Miss JOSHUA thanked the speaker for his masterly address, which she had found most stimulating. After reading Innes' review in *The Veterinary Record* and hearing Mr. Weiper's paper that day, one was rapidly coming to the conclusion that one had rarely, if ever, seen Laidlaw Dunkin canine distemper. She agreed with Mr. Weipers that pooling of clinical knowledge was likely to be most helpful and she therefore proposed to describe "hard pad" disease as it was occurring in her practice area rather than to discuss Mr. Weipers' remarks in detail. Her practice area was in North London: it was mainly suburban and comprised almost entirely single dogs kept as household pets. There was undoubtedly marked variation in the virulence and morbidity of the disease in different localities. In her area, where hard pad disease had been epidemic for about one year, it had at first been relatively mild in type, with a mortality rate of about 15 per cent., but in the last six to eight weeks there had been evidence of a distinct increase in virulence. She felt fairly certain that there existed a large number of chronic and even sub-clinical cases which never showed clinical symptoms, and considered that these were responsible for much spread of infection. She believed the disease to be insidious in onset, and although opportunities for routine temperature checking were few, she was of the opinion that there was no characteristic temperature rise at any stage. The incubation period in practice appeared to be some three to four weeks. She agreed with the Beckenham team that there was some "other factor" in the production of clinical manifestation of the disease, but had no idea what it was; she could not tie it up with any dietetic factor. She had wondered if the density of dog population walking over a given area had any influence in the spread and virulence of infection. If so, this might account for the peculiar street-by-street spread she had noticed, and also the large number of outbreaks after a big show, due to the concentration of dogs in the show rings. It might also account for the difference in severity of outbreaks in different kennels, *e.g.*, where hygiene was good and individual runs the rule, the mortality rate was relatively low, but in overcrowded kennels using a common exercise run, losses were very high. The enlargement of tonsils described by the Wellcome team was present in a high proportion of cases at one stage or another; it seemed to her that rather than an active tonsillitis there was an oedematous condition of the tonsils and crypts which showed itself as a translucent engorgement of the tonsil. The heart appeared frequently to be involved and she had noted that there was often no correlation between the strength of the pulse and that of the heart beat, while Miss Freak had observed that the characteristic finding on auscultation was a blurring or muffling of the first heart sound. In many cases the dog's expression changed; there was often an anxious expression reminiscent of a threatened peritonitis, and in some cases there was wasting or displacement of the temporal muscles—she had the impression that many of the dogs had severe headache. Photophobia was a common symptom early in the disease and when it occurred she regarded it as almost diagnostic. Oculo-nasal discharge in most of her cases had been mucoid or muco-purulent rather than watery, as described by Mr. Weipers, with occasionally a copious, purulent discharge of the eyes, usually associated with *bronchosepticus* infection. In spite of the fairly frequent occurrence of respiratory symptoms she had not seen a single case of pneumonia; the symptoms in her area were an intermittent dyspnoea, particularly occurring in the evening, that she believed to be due to a transient pulmonary oedema, but whether this was cardiac or C.N.S. in origin she had no idea. Coramine by mouth relieved these symptoms. In a few cases she had seen, early on in the disease, a generalised twitching of the whole forequarter, a palsied condition not unlike the Parkinsonian syndrome in man. The dogs usually recovered from this phase. In one of her cases a dog, in spite of having made an apparent clinical recovery, ran a persistently high temperature for six to eight weeks, and on discussing this with Mr. MacIntyre he had suggested that it might be due to brain damage around the thermogenic centre; she had also had cases showing very acute and intractable vomiting in spite of recovery in other respects and she thought this might likewise be due to damage near the vomiting centre—these cases responded in a spectacular manner to chloretone. She had had two cases which had become hyper-acute, one with renal and one with gastro-enteric symptoms. Both dogs had collapsed to such an extent that destruction seemed the rational course; both, however, had been left and both had made uninterrupted recoveries from this point. Apparently it was a form of "shock" therapy. (Since the meeting a third case of this type has been seen with severe vomiting, dehydration and coma; at the time of writing the dog is recovering well.) She agreed with Mr. Weipers that hard pads occurred in only a proportion of cases, but she disagreed with the Beckenham team's view that pad lesions appeared after about one week; in her cases pad lesions often did not appear for five to eight weeks. The differences she had noted between her present outbreak of "hard pad" disease and that she had previously been taught to call canine distemper were:—

1. The primary tonsillar enlargement as opposed to the pharyngitis in Laidlaw Dunkin distemper.
2. The absence of any characteristic temperature rise.
3. The long incubation period.
4. The occurrence of infection in very young puppies, presumably signifying ante-natal infection. In her area suckling puppies showed diarrhoea and acute pad lesions and not the convulsions described by Mr. Weipers.
5. The hyperkeratitis. She could often note this earlier in the nose than in the pads.
6. The protracted course of the disease.
7. She thought there was some difference in breed susceptibility. Whereas the cocker spaniel was highly susceptible to the nervous after-effects of distemper it seemed to stand up farly well to "hard pad" disease; on the other hand, the distemper-resistant oriental breeds were quite susceptible to "hard pad."

She wondered whether there was any obscure immunological relationship between the two diseases, preventing their co-existence, since looking back over the past four years she was satisfied that "hard pad" was gradually replacing Laidlaw Dunkin in her area. Could this be due to the replacement of one virus by another? She asked if Dr. Montgomerie could give any details of the histopathology of various tissues, since "hard pad" seemed to leave far

more extensive tissue damage than Laidlaw Dunkin, and she had the impression that the damage might differ from the ordinary type of inflammatory reaction. She had some slight evidence of sterility in both dogs and bitches following outbreaks of "hard pad"; she had also seen abortion and other interferences with pregnancy and parturition associated with it which postulated damage to the endometrium and other organs of reproduction.

Miss Levie said that her practice was in the West Country and comprised city, suburban and truly rural. She recognised her first case of "hard pad" only last July and had had mild cases with a good percentage of recoveries even in cases where the foot lesions were marked. She queried Miss Joshua's remarks on the method of spread of the disease as she had had cases in most isolated districts. With regard to the susceptibility of the oriental breeds she had found that chows, at least, recovered.

Miss Milnes, of Huddersfield, described her practice as one in an industrial city with surrounding villages, and cited the case of a kennel of chows which came up from the South. Within a week two developed very painful hot feet, followed in a week by hardening of the pads and then sloughing. Temperatures ranged around 104° F. There was photophobia and a pneumonia similar to that caused by *Br. bronchosepticus*, followed by fits, but the dogs recovered. These were the only two cases she had met with that syndrome. She had met, however, two sets of symptoms—one, which she considered Laidlaw Dunkin distemper, with cough, tonsillitis, pharyngitis, diarrhoea, temperatures around 102° F. to 105° F., and a pneumonia responding to treatment unless symptoms of encephalitis supervened. The second followed a similar course with cough, tonsillitis, diarrhoea, and a rise in temperature, but the dog then recovered except for a temperature fluctuating between 102° F. and 103° F. for weeks, later becoming listless and followed by pneumonia like that caused by *Br. bronchisepticus*, but no photophobia and the pads soft. The pneumonia proved resistant to all treatment and within a week the pads and nose hardened, and then encephalitis always appeared unless "hard pad" serum was used. The long-drawn-out nature of these symptoms, with very late hardening of the pads and noses, seemed to her to be the only clinical difference between the two diseases. Consequently she felt that she gave "hard pad" serum far too late in many cases, as in view of its price and dosage she felt unjustified in using it without proving the disease, but Mr. Weipers had mentioned hysteria at the commencement of the disease being a diagnostic feature and she hoped, if this were so, to be able to get her serum in earlier in some cases. In the past six months she had had no inoculated dog go down with "hard pad" except the following case. She had attended one client with two Japanese spaniels and a Shetland sheepdog; the latter had been treated by her six months previously for distemper, one Japanese spaniel had been inoculated by the L. and D. method, but the other, a puppy of six months, had been taken south to kennels and within a week of its return showed a temperature of 105° F., conjunctivitis and tonsillitis. Caniseran and sulphatriad were given and the dog was normal in four days. Distemper serum was administered to the other two dogs on the owner's request, and a week later the Shetland developed diarrhoea but no temperature, and this responded quickly to phthalylsulphathiazole. The other Japanese spaniel ran a temperature of 104° F. with marked photophobia but no purulent discharge, but finally recovered after three weeks. The puppy died of chorea in a fortnight but there was no hardening of the pads. She presented statistics of cases treated and suggested that there seemed some evidence of breed susceptibility to death from encephalitis, as all the deaths were from this cause. Percentage of deaths from encephalitis:—Collie and cur collie, 75; alsatian, 70; spaniel, 50; wire and smooth fox terrier, 45; mongrel, 20. She dealt with many Staffordshire bull terriers but had never known a case in that breed, attributing this to the fact that the Staffordshire had been bred for stamina rather than for show points or high intelligence as in sheepdogs. She felt that the highest incidence appeared to occur in highly strung and intelligent breeds, and wondered whether close in-breeding for show points might increase the neurotropic tendency of the virus, as the incidence of encephalitis had increased markedly in late years, and so had the craze for pedigree dogs and the fixing of certain show points by close in-breeding.

Mr. W. R. Conn, of Kent, cited various cases which had shown the typical thickening of the pads and suggested that differential diagnosis in the early stages was very difficult. He suggested that of the many hundred dogs inoculated by the V.V method only a very small proportion had "gone wrong." He had noticed no breed susceptibility.

Miss James had an urban practice in Cardiff and most of her cases appeared to be of the Laidlaw Dunkin distemper. She had had no case in an inoculated dog. She agreed that in-breeding and over-breeding of a particular breed would increase its susceptibility to nervous symptoms, and mentioned the corgi in her district where 40 per cent. to 50 per cent. developed these.

Miss Woods said that her practice was mostly country or country towns in Yorkshire and mentioned two features she had noticed consistently in her "hard pad" cases. First, a very heavy scurf on the head and down the sides, often developing before any sign of malaise, and, secondly, a consistent, high temperature of 103° F. lasting for weeks. She had not yet seen a case where the nose hardened, and found that the nerve symptoms developed very late in the disease.

Miss Phillips, of Haslemere, said that her practice comprised country towns and villages and a "squatter's area." She found widespread "hard pad" disease in the overcrowded areas. She disagreed with Miss Joshua in that she found a very high initial rise of temperature of 105° F. to 106° F. in cases recorded by "trained owners." In her early outbreaks, about 1943, she had tonsillitis and nerve symptoms more severe than in her present cases. A feature of these latter was, however, a most severe diarrhoea together with a cough and photophobia. In her experience inoculated dogs might get "hard pad," but the nervous symptoms were only slight and recovery was almost invariable. In her present outbreak nerve symptoms were insidious in origin, with inco-ordination, ataxia and, in some cases, a permanent blindness. Suckling puppies generally died in convulsions within a few days when the bitch was affected. She had met only isolated cases of Laidlaw Dunkin distemper in the past few years. With regard to treatment, cases where the serum was used in the early stages had been very satisfactory, and even in cases where the temperature rose to over 105° F. she had had good results. She mentioned the acute skin lesion against which one could do nothing. She had kept these dogs under morphia or nembutal but on coming round they had still savaged themselves and had to be destroyed. She used liver extract and vitamin B intramuscularly. She suggested that there was great need for a concentrated and combined action between the laboratories and the practitioners.

Dr. Montgomerie said that he appreciated that the question of differential diagnosis between true distemper and "hard pad" disease was difficult and not uncommonly a cause of considerable concern to the practitioner. So far as distemper was concerned, Laidlaw and Dunkin had given a good definition of that disease, although it was appreciated that the practitioner frequently did not see the condition in its early characteristic phase. There had, as yet, been no equally good description of "hard pad" disease, largely because the laboratory had been unable to reproduce it in all its field manifestations. In fact, the picture which had been produced had been based largely on clinical material and histories provided by a considerable number of practitioners up and down the country. Until the laboratory had succeeded in reproducing the disease, and probably even thereafter, its description to differentiate it from distemper would remain somewhat vague. This point was well illustrated when one attempted to answer enquiries from practitioners as to the incubative period. While it could be said that the inoculated dog fairly regularly showed the first sign of illness some seven days after injection, the development of recognisable symptoms in field cases might not occur until three or four weeks after the dog had been exposed to infection. In fact, there was a growing belief that the period between exposure and recognisable illness was often quite prolonged and that the latter might occasionally appear with explosive suddenness, influenced by some external factor. It had been contended that true Laidlaw Dunkin distemper had largely disappeared from the country. Experience at the Wellcome Laboratories would tend to confirm this view although, of course, it must be remembered that they received selected cases. Recently the incidence of true distemper among this material had increased.

It appeared from some remarks made in the discussion that what had been written from the laboratory point of view had left the impression that "hard pad" disease was believed to be a new entity, whereas it was accepted as very possible that the condition had existed in this country for many years. Unfortunately the standard method which the Laboratories adopted when examining material for the presence of distemper virus had almost precluded the detection of "hard pad" virus. If the two ferrets injected had not shown distemper by about the twelfth day of observation they were then tested for susceptibility by the injection of known virus. The subsequent appearance of disease had been ascribed to the second injection, whereas in at least some cases it probably arose from "hard pad" virus injected with the original material. It was only when it was appreciated that this other virus did exist, and had a longer incubation period, that the method of diagnosis was altered to cover the possible occurrence of the new virus in the original material.

One of the most puzzling features of this disease was the failure to reproduce it at the Laboratories. Material known to contain "hard pad" virus had been injected into believed-susceptible dogs.

These dogs showed no more than a variable temperature reaction, and in no case had encephalitis developed, whereas a sample of blood taken from the injected dog had produced true "hard pad" disease in ferrets. Various hypotheses had been offered as an explanation, and some felt quite strongly that it was some interference with the "vitamin metabolism" which gave the virus the opportunity to produce its full destructive effects in field cases. This idea received some backing from the reports that the subcutaneous injection of large doses of vitamin B had proved of value in field cases.

Mr. PUGH confirmed Miss Woods' observations on the presence of scurf in cases of hard pad. He felt that the vitamin B complex was in some way involved with the etiology of the disease. His cases which were treated with "hard pad" serum had not developed nervous symptoms, and this line of treatment was eminently satisfactory. He had observed that many greyhound kennels did not suffer from the disease, and wondered whether this was because greater attention was paid to their diet. The administration of sulphathiazine did interfere with some part of the synthesis of vitamin B and would appear to predispose to nervous symptoms.

Miss UVAROV, after thanking the speaker, said that her practice was in Surrey, and suburban in nature. She disagreed with Miss Joshua about the susceptibility of the Oriental breeds to "hard pad" and cited the case of a chow she attended 18 months ago, before the advent of the serum. This dog at ten months' old developed chorea, hard pads and paralysis, but subsequently recovered. She felt that there were changing types of the disease; six months ago her cases were acute with high temperatures, tonsillitis and nervous symptoms developing rapidly, but now they showed subacute symptoms, long-drawn-out, less nervous trouble and might not develop the hard pads. She would like another name than "hard pad" to be given to the disease. Differential diagnosis was difficult, but through the ophthalmoscope she had noticed very minute haemorrhages on the fundus of the eye, which in the later stages remained as pigmentation. The heart sounds were muffled and irregular. She had found that some dogs developed "hard pad" some two or three months after L. and D. inoculation. She saw no cases in poor people's clinics, but there were many cases of L. and D. distemper. If it was agreed that "hard pad" did not occur in dogs inoculated against L. and D. distemper, should dogs recovered from "hard pad" be inoculated against L. and D. distemper? Had salmonella been isolated from any cases exhibiting the severe diarrhoea?

Miss FREAK said that in a number of recovered or late cases of "hard pad" she had found small papules inside the rectum. They rarely caused symptoms, but if they did so they were similar to those of impacted anal glands. She would stress, however, that these were only found in late or recovered cases.

The Reply

Mr. WEIPERS, in replying, said that he had not noticed the papules in the rectum, mentioned by Miss Freak, but he frequently found pustules in various parts of the body in "hard pad" disease and these often set up an irritation which started the dog savaging itself. In severe cases it would even gnaw through its own bones. He had not recently demonstrated salmonella in bowel catarrhs but that was probably because of late he had not made many bacteriological examinations in such cases. About twelve years ago Prof. Dalling had isolated salmonella from material sent out by him for investigation. At the present time, with existing evidence, it was difficult to be sure about cross-immunity with Laidlaw Dunkin distemper and "hard pad" infection. He would welcome a change of name from "hard pad" disease—to, say, epidemic encephalitis or epizootic encephalitis—because many cases in an outbreak did not develop "hard pad," and with some clients this could be a source of embarrassment. Contrary to Miss Uvarov's experience, he knew that it occurred in the clinic in Glasgow, and the records showed a high mortality: about 80 dogs out of less than 100. He indicated that in his area there were marked variations in virulence. In 1947 there had been almost 100 per cent. mortality, but by the spring of 1948 the disease ran a much milder course, to be followed by a reappearance of more serious symptoms later in the year. He had only recently looked for haemorrhages on the retina and had observed small extravasations dorsal to the optic papilla in the angle between the vessels. He had noted the scurfy condition of the skin described by Miss Woods. Concentrated liver extract had been used in some cases of chorea with some measure of success, and while it was not a specific the method was worthy of further investigation. He considered that the greatest need at the moment was a reliable diagnostic in the early stages and he hoped that the examination of bladder cells for inclusion bodies might help. Scrapings from the tongue and conjunctiva, suggested elsewhere, might prove to be useful. Miss Milne's cases rather appeared to suggest infection with Laidlaw

(Concluded at foot of next column)

Dunkin virus. He was interested in Miss Joshua's case with vomiting due to C.N.S. damage and he considered that in other cases of brain damage—cerebral apoplexy or shock in the dog—vomiting was an invariable feature and useful in differential diagnosis. He thanked the meeting for the honour conferred on him, an honour which he very much appreciated, and regretted that time forbade a more complete reply to the discussion.

CLINICAL COMMUNICATION

Testicular Neoplasm Associated with Sex Inversion in the Dog*

P. W. DAYKIN and R. H. SMYTHE

NOTTINGHAM

It has been known for many years that alopecia and sex inversion may be associated with the presence of certain clinically benign neoplasms in the testicles of the dog. A number of case reports have referred to ectopic testicles. We wish to record the clinical syndrome arising from neoplasms observed in normally descended testicles and therefrom to formulate the hypothesis, on purely clinical evidence, that the testicle may, if not at all times then at least in certain circumstances, produce an oestrogen.

The effects of injecting an oestrogen (or a synthetic oestrogen such as stilboestrol dipropionate) in repeated and regulated doses into a normal healthy dog are constant and follow in regular sequence. Briefly, there is first a loss of hair of the muzzle, particularly noticeable in soft-coated terriers bearing a "moustache," followed by alopecia of the rump, thighs and abdomen, sometimes but by no means always, associated with pruritus. The skin of the lower abdomen takes on a slaty-blue pigmentation. The prepuce swells and becomes pendulous, partly owing to enlargement of the mamma on either side, and lactation follows. At this time, the dog commences to attract other males (apparently through changes, partly odoriferous, occurring in the perianal sebaceous glandular secretion), behaves in every way as a bitch, adopts the same method of courtship, and invites other dogs to mount him.

It will be realised that this syndrome differs entirely from that resulting from senile atrophy of the testicles or from emasculation.

Although we have recognised two types of benign testicular neoplasm associated with alopecia, we have found that only one, which we will style Type B, gives rise to clinical signs of sex inversion. It produces a syndrome identical with that resulting from the repeated injection of an oestrogen.

Type A testicular neoplasm may be associated with senile atrophy or may arise independently at any time after the fifth year of life. On palpation and external observation, it is indistinguishable (with certainty) from Type B, but the alopecia arising from its presence, probably owing to destruction of interstitial cells, does not respond to the remedial treatment described later.

In both types there may be a palpable hardening of one or both testicles, accompanied usually by slight enlargement, although in some instances there may be some degree of atrophy. The former is more characteristic of Type B and the latter of Type A.

On section, the two types of neoplasm are readily differentiated. Macroscopically, Type A presents a uniformly grey surface. Type B shows a grey background, containing well-defined islands ranging in colour from chrome yellow to rose pink. The islands may be oval or circular and discrete, each about 2 mm. in diameter, or may later coalesce into one or two kidney-shaped masses, occupying the greater part of the testicular substance. They do not invade either the epididymis or the tunica albuginea. Microscopically, Type A neoplasms show a degeneration or complete absence of spermatogenic cells with a marked invasion of fibroblasts.

In the case of Type B, microscopical examination of a section of "island" shows that the spermatogenic cells have disappeared completely and that varying numbers of adjacent seminiferous tubules have become confluent, leaving circular spaces of irregular diameter, occupied and sometimes completely filled with cells which we believe to be Sertoli cells. The tissue surrounding the spaces carries a framework of fibroblasts, containing clusters of interstitial cells, readily distinguishable from the cells of the neoplasm, which radiate outwards from the fibrous walls of the cavities, their nuclei fusiform and their long axes perpendicular to the walls.

The question now arises as to the origin of the factor, presumably an oestrogen, responsible for the association of neoplasms, containing such islands, with the clinical picture of sex inversion. On purely hypothetical reasoning, we suggest that either Sertoli cells

* Summary of Communication made to the East Midlands Division, N. V. M. A., at Sutton Bonington, June 17th, 1948.

or (possibly) interstitial cells may, in certain circumstances, produce an oestrogen which normally would be balanced or antagonised by the secretion of an androgen from a healthy testicle. What happens in such a case if the secretion of androgen be restored or stimulated?

The method adopted by the writers, in 16 cases, has been the subcutaneous injection at weekly intervals of 250 I.U. chorionic gonadotrophin (luteinising hormone) with the object of stimulating the interstitial cells to produce testosterone. The results have been spectacular. After the third injection, the prepuce has returned to normal size and lactation has ceased. Skin pigmentation has disappeared, long hair has grown on the muzzle and the alopecia has been replaced by a dense growth of hair. The dog now snaps at other males and at the end of the sixth week of treatment, has regained, so far as his age may permit, all his former masculinity.

We are unable to state at present the duration of this state after the cessation of treatment but we have records of two cases in which the normal sex characteristics have persisted for six and nine months respectively.

Our chief interest lies in the clinical evidence that stimulating the secretion of an androgen has apparently overcome a syndrome which we believe has arisen from the presence in the body of an oestrogen of testicular origin. There is no evidence that the testicular neoplasm is itself a product of oestrogenic stimulus, neither is there evidence that the cells producing the oestrogen may not be present in normal testicular tissue.

We have examined and sectioned the prostate in a number of cases of sex inversion associated with testicular neoplasm and in no case have we detected changes other than those associated with age.

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Trivia

Shock Therapy

I went to the meeting of vets. at —— yesterday. Most of the speakers were professors or research men, or veterinary inspectors who all of them have fixed and certain incomes.

To hear them solemnly discuss the use of most expensive inoculations was to me ridiculous, and when they had finished a Scot got up and said:

"It is all very interesting, gentlemen, but there is one thing you have all forgotten. I have to *make a living* out of my work."

The effect on this large gathering, some 110 doctors, vets. and research men, reminded me of the following.

Imagine the lady who holds spiritualist seances, with the room darkened and she at the end of the room solemnly pretending that the spirit is about to be made manifest. Everyone is keyed up, intense and attentive. Suddenly a loud hammering at the door.

Indignantly the spiritualist leader tries to appear unconcerned: still the knocking gets louder.

She has to go to the door. Then she sees a bedraggled maid, and she asks why on earth she has been interrupted.

"Lor', ma'am," shouts the slavey. "I 'ad to come that I 'ad to. 'Cos it's the landlord asking for the rent and he says that if I don't tell you he must have it at once he'll come up himself that he will."

What I mean is the sudden bringing the audience back to reality. I was very amused.—*From our Post bag.*

. . . . .

I am reminded of the man who was sitting at the Zoo when another man rushed up to him and said: "There's a moose loose," but who merely turned round and asked: "Are you talking Scots or English?"—Debate, Pests Control Bill.

Questions and Answers

The submission of questions for inclusion in this column will be welcomed; they can relate to any aspect of veterinary work. For purposes of record, each question is numbered, and those submitting questions are required to furnish (not for publication) name and address.

Answers to readers' queries represent exclusively the personal opinions of the writers, and criticism of the replies will be appreciated.

All communications should be addressed to the Editor.

"Knuckling Over" at the Fetlock Joints

Q. CCCV.—*It was noticed that a thoroughbred yearling filly was becoming very straight in the pasterns in both fore legs. A few weeks later she went very lame on the near fore leg. She had had a very bad cut on the outer aspect of the first phalanx about nine months previously. She was left with a thickening there, which according to the owner had increased recently. Quite a lot of pain was elicited on palpation of this swelling. Shortly afterwards she became lame on the other fore leg. She now "knuckles over" on both fore fetlocks, the near fore being the worst. She is now becoming straight on her hind pasterns also.*

Is this a nervous disorder due to mineral and vitamin deficiency accentuated on the near fore leg owing to previous injury? What treatment do you recommend?

A.—Cases of this sort are seen in debilitated animals but it is difficult to lay the blame for the condition on any particular dietetic deficiency. Young animals kept indoors and suffering from rickets may present symptoms closely resembling those described. The root cause is probably nutritional and if this is not due to serious circulatory disturbance from worm infestation improvement in the management and diet may be effective. Similar cases have shown remarkable improvement after spending a summer turned out in company in marshy pastures, the advantage being derived from sunshine, green food and plenty of exercise in soft going. Supplementary feeding is usually necessary. Cases of contracted tendons in older horses will improve under these conditions. The writer recalls a steeplechaser so knuckled over from contracted tendons that it was considered by him and by several other veterinary surgeons to be quite hopeless. The animal spent the summer turned out in some Hampshire water-meadows and was so much better that he stood up to training and was run very successfully in several hurdle races the next season.

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Summer Sores on Cows' Teats

Q. CCCVI.—*What is the treatment and cause of the raw, bleeding areas which appear on cows' teats during the summer? There also appears to be a discharge of serum and in one case lesions appeared under the belly and on the shoulder. Does the infection persist in buildings? In one case, where penicillin ointment proved ineffective, the skin was greatly thickened.*

A.—The lesions described may usually be attributed to infected fly bites. In the experience of the respondent, the following treatment has proved successful:—

1. The sores should be thoroughly cleansed with soap and warm water.

2. Sulphanilamide *c.* sulphathiazole cream (3 per cent. aa) applied at least twice daily.

3. The use of the long-lasting and reliable fly-repellents now obtainable should be suggested.

It should be emphasised that the apparent failures obtained with penicillin ointment are due to the instability of this preparation, and the fact that aseptic precautions must be employed in its application.

4. As an addition to the treatment, it may be necessary to tie the cow up in a stall so that she cannot reach the affected areas with her tongue, thus preventing healing by licking.

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Penicillin Medication and Cheese Making

Q. CCCVII.—*When cheese is being made at a local factory it is noted that the process in the vats containing milk from certain farms is appreciably slower than normal. Is there any justification for the suspicion that the use of penicillin for mastitis control on these farms may have a bearing on this?*

A.—There is very strong evidence to support the view that penicillin, by virtue of its influence on streptococcal organisms, has a marked influence upon the making of cheese. Its effect is in direct proportion to the strength of the penicillin which finds its way into the total mass of milk which is being used in one particular batch. If the milk from only one or two treated cows gets into the bulk at the factory, then it is unlikely to have any effect upon the cheese-making process. If one herd is mass treated and the milk from this is mixed with that of a small number of farms, then there may be some appreciable suppression of the growth of strepto-

cocci. This might well apply in the case of small factories receiving limited quantities of milk, or, of course, where the milk is split up into small lots of, say, 100 gallons before conversion into cheese. In this instance, the milk from a heavily treated herd might influence this particular batch. By and large, therefore, there is less likelihood of the process of cheese making being interfered with by penicillin at big factories where large volumes of milk are processed at one time. But where the supply is small the milk from a blitzed herd might well seriously influence the total supply of milk received for the day. The problem is at the moment being investigated in some detail at Reading University.

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Ear Cropping in Boxers

Q. CCCVIII.—*Is it legal, in this country, to crop the ears in boxers? If so, please describe the method of performing the operation.*

A.—There is no specific Act or Order which makes it illegal to carry out this operation. Attention is drawn, however, to the decision of a recent meeting of the Organising Committee of the Association, confirmed by Council on April 22nd:—

"That the cropping of the ears of boxers be strongly deprecated by the Association and that veterinary surgeons be advised against performing this operation."

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Correspondence

"Roaring" in Cattle

Sir,—As writer of the question No. CCC *re* "'Roaring' in Cattle," I wish to comment upon the reply given in *The Veterinary Record* of April 30th.

Spontaneous recovery is not the general rule. Of approximately 30 cases seen over the past two years, all were becoming worse until treated by blistering. Most cases when first seen had been in existence for periods up to one month. This fact, in my opinion, is against the possibility of the condition being due to urticaria. Udall states that the condition is *occasionally* a form of urticaria. Of the 30 cases seen only two failed to respond to blistering. So, under the circumstances, blistering may be regarded as an effective method of treatment.

Tracheotomy was performed on the two which did not respond to blistering and I maintain that this was not the policy of an alarmist. The operation had been postponed for some time until it became a question of operate or slaughter. Both animals were on the verge of asphyxiation! the visible mucous membranes were cyanotic, the tongue was hanging out and the animal was obviously breathing only with the greatest difficulty.

One operation case recovered and is now normal although the tube has not been used for 12 months. The other became progressively worse and was slaughtered. On *post-mortem* examination of this animal the larynx was found surrounded by what appeared to be a chondroma-like growth. Unfortunately, no laboratory report was obtained.

I think that the above points are sufficient to emphasise my contentions that (1) spontaneous recovery is not the rule; (2) the condition is rarely a form of urticaria; (3) blistering seems to be effective in the majority of cases, and (4) sometimes tracheotomy is essential to save the animal.

Yours faithfully,

87, Grove Road, A. M. Edwards.
Bridgend, S. Wales.
May 15th, 1949.

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Alopecia in Cats

Sir,—Your respondent is well aware of the school of thought which believes alopecia in cats to be due to hormone unbalance in neutered cats, but having seen the condition in a number of entire cats of both sexes, cannot accept this as being the sole cause. I am aware also, that good, if temporary, results are claimed for implantation of testicular tissue into neutered cats; thyroid, in my hands, has not given particularly encouraging results. It is surprising to learn that farm and other country cats are frequently castrates!

I agree with your correspondent that on enquiry into the diet of affected cats this often seems adequate, yet the fact remains that the number of such cases seen in practice has increased materially during and since the war, a period during which diet has undoubtedly been affected, yet the proportion of neutered to entire cats presumably remains the same.

There is evidence to show that biotin may be destroyed in the alimentary tract by certain proteins (egg white is one example) and it may well be that in some cases while the actual intake of biotin is adequate some other factor in the diet may be interfering with its utilisation and thus giving rise to clinical symptoms of deficiency.

Yours faithfully,
Respondent to Q. CCXCVII.

ABSTRACTS

[**Penicillin in Swine Erysipelas.** Aitken, W. A. (1949.) *N. Amer. Vet.* **30.** 25.]

The author briefly records the successful treatment of swine erysipelas with a combination of penicillin and erysipelas serum. Ten shotes ranging in weight from to 125 to 150 lb. and showing typical symptoms of swine erysipelas were back to normal and eating within 24 hours of being given a dose of aqueous penicillin (50,000 units each) along with 35 to 45 c.c. of erysipelas serum. Six of these had previously failed to show any improvement after a similar dose of the serum alone and one had died. The penicillin dose was repeated in eight hours. The author cites another case in which crystalline penicillin (dose from 300 to 1,000 units per lb. body weight) dissolved in erysipelas serum (2 to 3 c.c. per 10 lb. body weight) was used on 185 sick pigs from three to five months old in 18 herds. Mortality rate in acute cases was thereby lowered considerably compared with previous years, as was also the incidence of chronic arthritis among recovered pigs.

The author's remarks are in every case made with caution, but he suggests a dose of 1,000 units of crystalline penicillin in about ½ c.c. of erysipelas serum for each lb. of body weight, repeating the dose in 12 to 24 hours.

J. M.

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[**The Reaction of the Male Toad to Pregnant Mare's Serum and its Comparative Study with the Cole-Hart Test.** Neto, J. F. T. (1949.) *Amer. J. vet. Res.* **10.** 74.]

The author studied the reactions of male toads (*Bufo species*) to serum of mares, 36 to 111 days after service. Serum from mares which have conceived caused the extrusion of spermatozoa, due to the presence of circulating gonadotrophins. Portions of each serum sample were used for the rabbit ovulation test of Cole and Hart. Mares were frequently examined and complete records of gestation were available. The spermatozoa could be detected in the toad's urine which was easily obtainable from the cloaca. The urine was examined prior to testing to ensure freedom from spermatozoa, and 9 to 20 c.c. of serum was injected into the lateral lymph sac. The urine was then examined at 15 minute intervals from one to six hours, and then, if negative, at 12 hours and 24 hours following the last examination. The rabbit ovulation and male toad tests agreed in 36 of 39 cases (25 positive, eleven negative). Two cases agreed at a later testing and one was not re-tested. Toads can be used repeatedly, but require seven days' rest following a positive result. Occasionally toads died two to seven days after the test.

D. H. L. R.

NEW GRASSLAND RESEARCH STATION

Appointment of Director

Dr. William Davies, at present the Director of the Grassland Improvement Station of the Ministry of Agriculture and Fisheries at Drayton, Stratford-on-Avon, has been appointed Director of the Grassland Research Station for Great Britain, which is being established at Hurley in Berkshire and with which the Drayton Station is being amalgamated under the new Governing Body whose formation was recently announced.

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CALIFORNIA STATE VETERINARY MEDICAL ASSOCIATION

The above Association ask us to announce that their annual convention is being held on June 20th, 21st and 22nd, at the Chase Hotel, Santa Monica, California.

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Weekly Wisdom

You cannot have just a little State planning, any more than you can be just a little bit pregnant.—James S. Duncan, Canadian industrialist.

NATIONAL VETERINARY MEDICAL ASSOCIATION OF GREAT BRITAIN AND IRELAND

PROVISIONAL PROGRAMME OF ANNUAL GENERAL MEETING AND CONGRESS, 1949

	At Central Hall, Westminster (*Simultaneous translation available*)	At Church House, Westminster (*Consecutive translation available when required*)	
Friday, August 12th		Hoare Memorial Hall	Bishop Partridge Hall
12.15 p.m.	Annual General Meeting including Presidential Address.		
*1.15 p.m.	Luncheon interval.		
2.15 p.m.		*Paper*: "Organization of Veterinary Clinical Practice." By Mr. L. Guy Anderson (Aylesbury). Openers of discussion: Mr. E. R. Ambrose (Camberley), Mr. H. Raynor Hewetson (Southport).	*Paper*: "Clinical Aspects of Diseases of the Alimentary Tract of the Cat." By Mr. W. L. Weipers (Glasgow). Openers of discussion: Miss Marion Freak (Potters Bar), Miss C.A.Woods (Menston).
8.45 p.m.		*Paper*: "Physiology of Reproduction in the Dog." By Mr. I. W. Rowlands and Mr. J. L. Hancock (Frant). Openers of discussion: Mr. R. E. Williams (Southampton). and another.	*Paper*: "Urolithiasis in Dogs and Cats." By Dr. Agnete Krabbe (Copenhagen). Openers of discussion: Major Hamilton Kirk (London), Mr. J. S. J. Lauder (Altrincham).
8.30 p.m.	President's Reception and Dance, Dorchester Hotel, Park Lane, W.		
Saturday, August 13th			
10 a.m.		*Paper*: "Leptospirosis." By Miss J. O. Joshua and Dr. J. C. Broom (London). Openers of discussion: Dr. Agnete Krabbe (Copenhagen), Prof. A. Brion (Lyon, France), Mr. W. I. M. McIntyre (Edinburgh).	*Paper*: "Rickets and Tetany in Carnivores." By Dr. Arne Helgebostad and Dr. Nils Böhler (Oslo, Norway). Openers of discussion: Prof. A. N. Worden, Mr. Alan A. Wilson (Southport).
11.30 a.m.		*Papers*: "Advances in Bone and Joint Surgery in Dogs." (i) "Recent Experiences in Surgical Treatment of Fractures." By Dr. H. Moltzen Nielsen (Copenhagen). (ii) "Surgery of the Stifle Joint in Dogs." By Dr. Fritz Nilsson (Halsingborg, Sweden). Openers of discussion: Mr. Gordon C. Knight (London), Lieut.-Col. J. Hickman (Newmarket).	
*12.45 p.m.	Luncheon interval.		
2 p.m.	*Paper*: "Diseases of Household Animals Communicable to Man." By Dr. J. Winsser (Leiden, Holland). (To be presented by Dr. John Peters (Rotterdam).) Openers of discussion: Mr. Alex. Thomson (Glasgow), Dr. H. Williams Smith (London), Mr. L. K. Whitten (New Zealand).		
3.30 p.m.	*Paper*: "Techniques in Small Animal Surgery," illustrated with coloured films. By Dr. James Farquharson (Colorado, U.S.A.). Openers of discussion: Mr. G. N. Gould (Southampton), Mr. S. F. J. Hodgman (Newmarket).		
Evening.	Various theatre parties organised by Ladies' Veterinary Benevolent Guild.		
Sunday, August 14th.	Free day.		

Royal Veterinary College, Camden Town

***Monday, August 15th.** Demonstrations throughout day. Full details will be announced later but will include the following:—

Paper: "Surgery of the Ears in the Dog," illustrated with coloured films. By Dr. C. P. Zepp, Senr. (New York) (President of the American Veterinary Medical Association). Openers of discussion: Prof. C. Formston (Streatley), Mr. C. E. Woodrow (London).

Talk: "Fracture Repair by Bone Pinning," illustrated with coloured slides. By Dr. Jack O. Knowles (Miami, Florida).

Talk: "Intratracheal Narcosis in Small Animals," illustrated with film. By Dr. E. J. Voute (Amsterdam).

Demonstration of Osteosynthesis. By Dr. H. Moltzen Nielsen (Copenhagen).

3.30 p.m. Adjourned Annual General Meeting (Members only), Grand Hall, Royal Veterinary College.

4.15 p.m. Closing Meeting, Grand Hall, Royal Veterinary College

5 p.m. (approx.). First Meeting of Council, N.V.M.A., for 1949-50 (Members only), Grand Hall, Royal Veterinary College.

* Luncheon and Tea will be available on the cafeteria system at the Central Hall and at the Royal Veterinary College.

NOTES AND NEWS

Diary of Events

June 7th and 8th.—D.V.S.M. Written Examination, Royal (Dick) Veterinary College, Edinburgh.

June 8th.—Annual Meeting of the West of Scotland Division, N.V.M.A., at Glasgow (North British Hotel), 2.30 p.m.

June 8th.—Meeting of the Midland Counties Division, N.V.M.A., at Birmingham (Grand Hotel), 2.30 p.m.

June 9th.—Annual General Meeting of the Research Defence Society, Manson House, 26, Portland Place, W.1, 3.15 p.m. (Stephen Paget Memorial Lecture: Professor C. Lovat Evans—"Physiological Research and the Vivisection Act.")

June 9th.—Joint Summer Meeting of the Sussex and South-Eastern Divisions, N.V.M.A., at Frant (Veterinary Research Station, Ely Grange), 2.30 p.m.

June 10th.—Annual Dinner of the R.A.V.C. Officers' Club (Grosvenor House, W.).

June 13th.—Emergency Meeting of Lincolnshire and District V.M.A., Institute of Animal Pathology, Milton Road, Cambridge at 2.30 p.m.

June 15th.—Annual General Meeting of Lincolnshire and District V.M.A., at Saracens Head Hotel, Lincoln, 1 p.m.

June 15th.—Meetings of the N.V.M.A. Editorial Executive Committee, at 36, Gordon Square, W.C.1, 2 p.m.; Main Committee, 3 p.m. (Note alteration from 2nd to 3rd week in the month).

June 16th.—Summer Meeting of the Lancashire Division, N.V.M.A., at the County Institute of Agriculture, Hutton, nr. Preston.

June 17th.—Meeting of the Biochemical Society at Nottingham (Department of Biochemistry, the University) 2 p.m.

June 22nd to 24th.—June Meetings of N.V.M.A. Council and Committees in Edinburgh:

June 22nd.—Organising Committee, 2 p.m.; Home Appointments Committee, 4 p.m.

June 23rd.—Veterinary State Medicine Committee, 10 a.m.; General Purposes and Finance Committee, 2 p.m.

June 23rd.—Meeting of Western Counties Division, Royal Clarence Hotel, Exeter, 2 p.m.

June 24th.—Parliamentary and Public Relations Committee, 10 a.m.; **Meeting of Council,** 11.30 a.m.

June 21st to 24th.—Royal Highland and Agricultural Society's Show at Dundee.

June 23rd and 24th.—R.C.V.S. Membership Written Examination.

Aug. 8th to 13th.—Fourteenth International Veterinary Congress, Central Hall, Westminster, and Church House, Westminster, London, S.W.1.

Aug. 12th to 15th.—Sixty-seventh Annual General Meeting and Congress, N.V.M.A., in London.

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R.C.V.S. Council Election Result

The following result of the ballot of members taken for the election of 20 members of Council was declared at the annual general meeting of the Royal College of Veterinary Surgeons, held at 10, Red Lion Square, London, W.C.1, on Wednesday last:—

Term of Office	*Elected*		*Retire at Annual General Meeting in*
4 years	1 Montgomerie, R. F.	1,252	1953
	2 Mitchell, T. M.	1,182	
	3 Anderson, L. Guy	1,120	
	4 Pugh, L. P.	1,103	
	5 Edwards, J. T.	1,067	
3 years	6 Gould, G. N.	1,067	1952
	7 Barker, J. R.	1,066	
	8 Sumner, H.	1,062	
	9 Wooldridge, G. H.	1,059	
	* *Glover, R. E.*	1,035	
	10 Boddie, G. F.	1,012	
2 years	11 Male, G. P.	992	1951
	12 Procter, J. W.	989	
	13 Bosworth, T. J.	970	
	14 Wooldridge, W. R.	968	
	15 Steele-Bodger, H. W.	940	
1 year	16 Emslie, J. W.	912	1950
	17 Peele, J. D.	910	
	18 Tutt, J. F. D.	884	
	19 Share-Jones, J.	876	
	20 McKinna, W. R.	874	

Not Elected

21	Spicer, A.	868
22	Brown, E.	846
23	Motton, S. J.	838
24	Carmichael, J.	834

* Although elected as above, Professor Glover having been appointed a representative of the University of Liverpool, has surrendered his seat as an elected member with consequential alteration in the term of office of some members.

It will be noticed that there is a tie for fifth and sixth places. In accordance with section 11(3) of the Veterinary Surgeons Act, 1948, Dr. Edwards has been allotted the fifth place as having been first entered on the Register of Veterinary Surgeons before Mr. G. N. Gould who is allotted sixth place.

. . . . . .

The following are the *appointed* members of the Council:—

Privy Council	P. H. Collick, Esq., M.P. W. McNair Snadden, Esq., M.P. Professor T. Dalling H. W. Dawes, Esq.
Universities (in alphabetical order):—	
Bristol	Professor F. Blakemore
Cambridge	„ W. I. B. Beveridge
Edinburgh	„ F. A. E. Crew
Glasgow	„ R. C. Garry
Liverpool	„ J. G. Wright „ R. E. Glover.
London	J. B Hunter, Esq.

Affiliated Veterinary Colleges (in alphabetical order):—

Royal (Dick) Veterinary College, Edinburgh	Professor W. M. Mitchell
Glasgow Veterinary College	„ A. A. Forsyth
Royal Veterinary College and Hospital of London	„ J. B. Buxton

REPUBLIC OF IRELAND

Nominated Member

Nominated by Ministry of Agriculture of the Republic
Prof. T. G. Brown.

Elected Members

P. F. Dolan.
Prof. W. Kearney.
P. E. Mullaney.

By reason of the nomination of Prof. Brown, there is one vacancy for an elected member.

The New Council of the Royal College

The new Council of the Royal College of Veterinary Surgeons, whose composition was announced at the annual general meeting on Wednesday, June 1st, represents a development of the greatest importance in the history of the veterinary profession.

Since the first Royal Charter in 1844 the Council has consisted of elected members. The original Charter provided for not less than 24 and not more than 31. This number was altered by the Royal Charter of 1892 to be 32. Following the establishment of the Irish Free State and the agreement entered into between His Majesty's Government in the United Kingdom and the Government of the Free State, the number was increased to 36 by the addition of four Irish elected members under the Royal Charter of 1932 and by the Veterinary Surgeons (Irish Free State Agreement) Act, 1932. From that time until this week the members have remained at 36, all of whom are elected members.

Section 9 of the Veterinary Surgeons Act, 1948, laid down that at the first annual general meeting held after the commencement of the Act all the existing members of the Council should vacate office and as from that meeting the Council should be reconstituted in accordance with the provisions of the Act. These provisions lay down that there shall be 20 elected members who are members of the Royal College and who are elected by their fellow members residing outside Eire (now the Republic of Ireland). It is further laid down that there shall be four persons appointed by the Privy Council and two persons for each University in the United Kingdom which has a registrable veterinary degree. Of the two representatives from each University one must be a member of the Royal College.

The Act, however, laid down certain transitional provisions pending the establishment of a registrable veterinary degree in the various Universities. It is under these transitional provisions that the appointments listed above have been made.

The representation of the Republic of Ireland is the result of an agreement which was signed very recently by the High Commissioner for the Republic of Ireland in London, Mr. Dulanty, with the Secretary of State for Commonwealth Affairs, and which was given effect to by an order in Council just before the annual general meeting. We believe that this agreement is the first to be made between the United Kingdom and the Republic of Ireland since the recent constitutional changes.

The Council in its new form follows the lines already established in the case of the General Medical Council, the Dental Board and other similar professional bodies. It is the outward form of a new era in veterinary education in this country. The new members will be welcomed by the veterinary profession in the hope that they will contribute to the benefit of, and wise guidance of, the profession and whilst there have been differences of opinion in the past as to the implementation of the Loveday Reports, it is hoped that every member of the profession will give the new Council the fullest support in the future.

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XIVth International Veterinary Congress

The Belgian Delegation

We are indebted to Professor Verstraete, Principal of the Ghent Veterinary School, for the following particulars regarding the members of the Belgian delegation at the XIVth International Veterinary Congress to be held in London in August.

The delegation will comprise:

L. Geurden, VET.D., professor of hygiene and bacteriology, Director of the clinic for poultry and small farm animals, who will present a paper, "Botulism in Men and Animals" (Section 7A). Professor Geurden is also the official delegate of the Minister for National Education of Belgium.

G. Peeters, VET.D., professor of physiology, pharmacology, pharmacodynamic toxicology, presents a paper, "Physiology of the Udder" (Section 4E).

M. Vandeplassche, VET.D., professor of obstetrics and gynaecology, Director of the Centre for Artificial Insemination, Ghent, presenting a paper, "Viability of Bull Semen in the Genital Tract of the Cow" (Section 5D).

J. H. Bouckaert, VET.D., professor of veterinary surgery, Director of the clinic for large animals.

A. E. R. Willems, VET.D., professor of zootechny and agricultural husbandry and economics.

R. Vercruysse, VET.D., assistant in the laboratory for parisitology.

(All the above are members of the staff of the Veterinary School of Ghent.)

Ch. Van Goidsenhoven, VET.D., Rector of the Veterinary School of Cureghem (Brussels), professor of bacteriology.

G. Degryse, VET.D., Head Inspector and Director at the Ministry of National Health and of the Family, will represent the Minister of that Department.

Both Veterinary Schools of Belgium—Ghent and Cureghem—have entered a subscription as ordinary members of the Congress.

Mesdames Geurden, Peeters, Bouckaert, Willems and Vercruysse will accompany their husbands and have subscribed as associated members of the Congress.

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PERSONAL

Birth.—PRITCHARD.—On May 29th at the Denbighshire Infirmary, to Margot, wife of Robert W. Pritchard, M.R.C.V.S., of Plas Coch, Denbigh, a son.

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R.C.V.S. OBITUARY

GOODWIN, Harry, Antigua, British West Indies. Graduated London, March 21st, 1887. Died December 8th, 1947. (Information just received.)

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Necropsies and Tuberculous Infection

"The risk of contracting tuberculosis at *post-mortem* examinations, both for participants and onlookers, may have been underestimated," observes the *British Medical Journal* in its issue of May 21st, and continues: "At Lund University, as elsewhere in Sweden, medical students attend a general pathology course, which includes necropsies on tuberculous cases, before they start their regular clinical work. Prior to wholesale B.C.G. vaccination many of the students were tuberculin-negative on entry, and the Students' Tuberculosis Bureau, by analysing figures of conversions from tuberculin-negative to positive and of tuberculosis morbidity, was able to show an association between these and attendance at the pathology course, quite apart from any association with the period of clinical training.[1,2] Direct bacteriological examinations showed that many objects in the *post-mortem* room were contaminated 24 hours after examination of the body of a tuberculous patient, in spite of routine cleaning. Following the introduction of stringent precautions at such necropsies there was a decrease in the tuberculin-test conversion rate among the students attending the pathology course. Among these precautions were exclusion of students from actual participation in the *post-mortem* examinations of known tuberculosis cases, the fitting out of a special necropsy room with improved cleaning facilities, and injection of formalin into the pulmonary artery of the cadaver.

"More recent evidence from the University of Rochester medical school in the U.S.A. amplifies the Swedish experience.[3] At this medical school, where there was a high tuberculin-test conversion rate and a high tuberculosis attack rate among the students, tuberculin-testing revealed that most of their primary infections occurred during the second year, when a 20-weeks pathology course was taken, but before the practical clinical work was started. All these students had opportunities for coming into frequent contact with tuberculous material. At the end of the first year an average of 54 per cent. of the students were positive reactors, and the figure rose to 92 per cent. at the end of the second year. After exclusion of second-year students from participation in the necropsies of tuberculous cases and from the handling of tuberculous tissues it was found that on an average 36 per cent. of the students were positive reactors at the end of the first year, 39 per cent. at the of the second, and 52 per cent. at the end of the fourth year.

"In British medical schools the students usually do not attend *post-mortem* examinations regularly until their clinical course has

begun, and it would be difficult to assess the separate danger of developing a tuberculous infection as a result of taking an active part at such examinations. However, bacterial spread around the cadaver and *post-mortem* table should be investigated in order to identify the most risky procedures. Meantime, though the Scandinavian and American results do not constitute proof, they provide grounds for revising the general opinion that the rapid conversion of medical students from tuberculin-negative to tuberculin-positive during their hospital studies, as well as their liability to contract tuberculosis, is solely the result of their contact with living patients. Tuberculous necropsy material must now be regarded as a source of infection, and all possible steps should be taken to reduce the risks to which medical students may be exposed. Few medical schools in this country can claim to conduct *post-mortem* examinations according to a rigorously strict hygienic routine. The adoption of operating-theatre processes (partially in reverse) should be considered: for the participants, masks (and, of course, gloves) and no smoking on the job; for the onlookers, a glass screen, care in the display of tuberculous viscera when removed from the body, and the application of disinfectants to supplement the customary hosing down with water afterwards. Such precautions should be supported by periodic X-ray examination of the students and by tuberculin-testing—procedures which still await full application in some medical schools.[4]

"Quite recently a new danger arising from tuberculous tissues has been reported, this time involving a large section of the public. There is little meat for pets in household scraps, and uncooked beef from tuberculous carcases unfit for human consumption is finding its way from knackers' yards to pet-food stores.[5] The unsuspecting or uninformed purchaser may contaminate her kitchen and endanger the health of her family. Moreover, cats and dogs can contract tuberculosis from eating raw infected meat. The National Veterinary Medical Association has given warning of these risks and has suggested that all meat sold at pet-food stores should be sterilised by boiling before sale.[6] In Birmingham the Corporation has obtained powers to enforce sterilisation."

REFERENCES

1 Hedvall, E., *Amer. Rev. Tuberc.* (1940.) **41.** 770.
2 Lindau, A., *Nord. Med.* (1941.) **9.** 858.
3 Meade, G. M., *Amer. Rev. Tuberc.* (1948.) **58.** 675.
4 Sixth Annual Report, 1947-48, of the British Medical Students' Association, London.
5 Montgomerie, R. F., *Lancet.* (1948.) **2.** 511.
6 *Lancet.* (1949.) **1.** 78.

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the N.V.M.A.

Correspondents are requested to write as briefly as possible.

HARD PAD DISEASE

Sir,—I congratulate Mr. J. K. Bateman on his excellent article in the *Vet. Rec.* No. 21. Vol. **61.** I must confess to being gratified to meet with part confirmation of my findings. (*Vet. Rec.* **61.** 163.) In particular it seems impressive that Mr. Bateman's group of 62 Wellcome Vaccination "non-reactors" showed a morbidity rate of 100 per cent., whereas my group showed a morbidity of 95 per cent.

It would certainly be helpful if the Wellcome staff would publish an authoritative explanation of the enormous variation in reactions following distemper virus injection. I am not aware of any other reasonably analogous biological, having been subjected to the standardisation as practised by the Wellcome laboratories, which gives rise to such variable results.

The paper by MacIntyre, Trevan and Montgomerie (*Vet. Rec.* **60.** 638) conflicts very strongly with Mr. Bateman's conclusions—especially para. 8 and 9 thereof, wherein he suggests that the whole epidemic was nothing more than distemper, in spite of the vaccine-virus inoculation, and the biological diagnosis.

He disputes, that hard pad virus is an entity. This conclusion overlooks many findings of MacIntyre, etc.

(1) The incubation period of hard pad virus is quite distinctly different from that of distemper virus.

(2) Ferrets injected with hard pad virus commonly recover. Ferrets injected with distemper virus rarely recover.

(3) Dogs (if susceptible) merely suffer a 10 to 14 day period of febrile indisposition on hard pad virus injection. Contrast this with the course taken by a dog injected with distemper virus.

(4) A hard pad immune dog is susceptible to distemper virus. A distemper immune dog is susceptible to hard pad virus.

(5) Hard pad virus is not neutralised *in vitrio* by hyper-immune distemper serum.

The clinical picture of hard pad and distemper are less clearly differentiated, but will certainly sustain the possibility of there being two diseases. Evidence is accumulating that distemper immunised dogs rarely exhibit pad hardening when subsequently afflicted with hard pad disease.

It has occurred to me that a batch of Wellcome distemper virus which causes a reaction may have been contaminated with hard pad virus—how else can we explain the subsequent resistance or immunity of dogs on which such a batch of virus was used. Viewed conversely the idea still holds good. No "reation," then no immunity if hard pad should break out.

This is dependent on the fact that a dog injected with hard pad virus is subsequently immune.

Such a suggestion, is I realise, futile unless the Wellcome Research Laboratories support or refute it on supportable grounds, and I have no doubt that it has occurred to them.

Yours faithfully,

79, St. Helens Road, F. G. Greer.
Leigh,
Lancs.
May 31st, 1949.

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PERSONNEL OF THE PROFESSION

Sir,—The discussion provoked by the letter of Professor McCunn raises questions of great importance to the veterinary profession.

The immediate problem is the requisite total annual output of veterinary graduates by the several schools. We can only attempt to solve this when we consider to what extent the Government intends to utilise veterinary services. Miss Ford would like to see veterinary science employed on the fullest possible scale, but Mr. J. Tweedale Edwards argues that such a state of affairs could only occur under a totalitarian régime, in which case, he believes the standard of living would be depressed. It is not clear what he means by "totalitarianism," but presumably he has in mind the type of dictatorship which prevailed in pre-war Germany. Here, undoubtedly, the general standard of living deteriorated, although the armament manufacturers, who naturally supported the Nazis, did extraordinarily well out of the war preparations.

Fundamentally the standard of living of a community depends on: (*a*) its natural resources; (*b*) the labour power (including technical efficiency and organisation) of its workers; (*c*) the number of people in the community among whom have to be shared the fruits of production which result from the human labour expended on the country's resources.

As veterinarians, I believe our main national rôle is to improve the efficiency of the stock-raising industry by promoting the health of farm animals. In this way, by increasing food production, we contribute to a raising of the standard of living. Quite clearly, the contribution which the profession can make to the "national cake" is proportional to the degree of application of veterinary science to animal husbandry. As a responsible national body, therefore, the N.V.M.A. should now be occupying itself with the construction of a system in which our services may be used to the maximum benefit of the community. Such thoughts may be unpopular in some circles, but if we are objective in our deliberations, we must face up to the organisation of veterinary services which the present era of food scarcity demands.

Yours faithfully,

Hilltop, G. H. Arthur.
Goring,
Reading.
May 17th, 1949.

* * * * *

FINANCIAL CONTROL IN THE NEW COUNCIL

Sir,—When the new Council meets it will be largely in the same position as the House of Lords and the House of Commons used to be as far as finance is concerned. There will be a number of members not appointed by the profession, some of whom do not even pay an annual fee, and yet they will have equal voice in matters dealing with finance with members elected by the profession.

In the near future the profession will have to find all the funds for carrying on the affairs of the college and for this purpose the

annual fee will have to be raised (the limit as fixed by the new Charter is five guineas) so it is only fair and democratic that the profession, through its elected representatives, should have the final say as to how the money should be spent.

Under the conditions that will now operate the only way of achieving this object, as far as may be, will be for the treasurer and all members of the finance committee to be selected from the elected members of Council.

In that case the profession could elect five new members to Council each year, if they were not satisfied with the financial policy; whereas if appointed members were included they would have little control.

Yours faithfully,

Oxted. ARNOLD SPICER.

* * * * *

PROFESSIONAL CONDUCT

Sir,—It is to be hoped that we are not to be treated to another spate of invective against our President and his Council and that the stream, which has appeared once more, may be staunched at once. In my humble opinion it is undignified.

One is disappointed that such a distinguished member as Mr. Steele-Bodger ignored or has forgotten Mr. Jorrick's advice to "count 20" before putting his views to paper, because his example may be followed by others of less importance.

Personally, I have always admired the forbearance of Council and their obvious reluctance to exact the full penalty of rustication if possible. Not lightly do they deprive a member of his bread and butter. Have the detractors of the Council ever thought "There but for the grace of God stand I"? Have they never erred themselves?

I can never understand why some members of the profession seem to enjoy shouting "stinking fish." It does the profession such a lot of harm both inside and outside the door.

Yours faithfully,

11a, Hamilton Road, R. H. C. HIGGINS.
Ealing, W.5.
May 21st, 1949.

* * * * *

THE FUTURE OF THE PROFESSION

Sir,—In my opinion no satisfactory reply can be given to Dr. McCunn's questions until it is known that, irrespective of what political party is in office, Chapter I of the "Second Report of the Committee on Veterinary Education in Great Britain" will be adhered to. This will entail, as it should, the removal of our agriculture from the lists of party strife, and its restoration to the position which it enjoyed before the repeal of the Corn Laws, which merely palliated a sharply defined agitation.

For a very long time, in quarters where attention might have been expected, and in other columns, I have stressed the urgency of the need to set up a Royal Commission to enquire into, and to report to His Majesty on, "The livestock economy of the British Empire," with adequate and fully informed veterinary representation on it.

The same train of thought would appear to be in the mind of Mr. Henderson, judging by his communication to these columns dated May 7th, when he stated, quite rightly, that our Colonial Veterinary Service should be entrusted with the development of the livestock industry in the colonies. For many years this branch of our profession has achieved far more than it has received by way of acknowledgment in return.

It is the veterinary profession alone, both here and elsewhere, that is capable of making it unnecessary to continue to haggle with a South American Republic over the supply and price of our meat, but all those who participate must receive the same remuneration.

Yours faithfully,

"Rothiemurchus," J. F. D. TUTT.
St. Cross, Winchester.
May 14th, 1949.

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HOSPITALITY FOR FOREIGN STUDENTS

Sir,—I request the courtesy of your columns for the following appeal.

The Association of Veterinary Students is endeavouring at present, with some success, to arrange for British students to see practice in various countries. To ensure the success of this scheme, however, we must be in a position to offer similar hospitality to foreign students visiting this country.

A number of students from abroad will be attending the International Veterinary Congress in London this summer and I would be pleased if we could invite a few of them to extend their stay and see conditions of practice in this country. Would any practitioners who might be able to offer such hospitality for a short period following the Congress please communicate with me at the following address.

Yours faithfully,

Students Representative Council, RONALD S. ORR
Glasgow Veterinary College, (President A.V.S.).
83, Buccleuch Street.
May 24th, 1949.

Conditions Governing Acceptance of Display Advertisements in "The Veterinary Record."

1. The Association cannot be regarded as approving the subject matter of any advertisements; nor is it a function of the Association to test the validity of advertised matter other than as outlined below.
2. The Association reserves the right to refuse publication of any advertisement without disclosing reasons to the advertisers.
3. Supply of prophylactic or therapeutic agents for veterinary purposes direct to the lay public may be regarded as adequate reason for refusing publication of any or all advertisements submitted by the firm concerned.
4. The general nature of prophylactic or therapeutic agents should be stated as part of the advertisement.
5. If any specific claims are made by the advertiser in relation to any product, they should be stated, briefly, or at length, as part of the advertisement and should be in reasonable accord with claims published elsewhere or commonly associated with the product.
6. In the case of new products, any claims of an unusual or important nature should be supported by positive evidence that the product is worthy of extensive field use.
7. If adequate evidence becomes available which indicates that claims made in respect of an advertised product are ill-founded, misleading or extravagant, reasonable notice will be given to the advertiser either to terminate publication in *The Veterinary Record* or to amend the wording of any claim made therein and elsewhere to the satisfaction of the Association.
8. "Copy" for all advertisements to be submitted for acceptance well in advance of publication (Press day: Wednesday), to the Editor, *The Veterinary Record*, 36, Gordon Square, W.C.1.

DISEASES OF ANIMALS ACTS, 1894 TO 1937, AND AGRICULTURE ACT, 1937 (PART IV).

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period	Anthrax	Foot-and-mouth	Fowl Pest	Parasitic Mange*	Sheep Scab	Swine Fever
May 1st to 15th, 1949	12	—	21	—	—	—
Corresponding period in—						
1948	3	—	12	—	3	—
1947	7	—	67	—	1	—
1946	3	1	—	—	—	20
Jan. 1st to May 15th, 1949	76	11	183	—	27	5
Corresponding period in—						
1948	43	—	173	1	26	13
1947	34	83	205	2	19	14
1946	44	23	—	4	34	238

NOTE.—The figures for the current year are approximate only.
* Excluding outbreaks in Army Horses.